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COMPRISING TEN VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY

UNDER THE GENERAL EDITORIAL CHARGE OF

GUSTAVUS P. HEAD, M. D.

PROFESSOR OF LARYNGOLOGY AND RHINOLOGY,
CHICAGO POST-GRADUATE MEDICAL SCHOOL

VOLUME X.

NERVOUS AND MENTAL DISEASES

EDITED BY

HUGH T. PATRICK, M. D.

PROFESSOR OF NEUROLOGY IN THE CHICAGO POLICLINIC; CLINICAL PROFESSOR OF NERVOUS DISEASES IN THE NORTHWESTERN UNIVERSITY MEDICAL SCHOOL; EX-PRESIDENT CHICAGO NEUROLOGICAL SOCIETY.

CHARLES L. MIX, A. M., M. D.

PROFESSOR OF PHYSICAL DIAGNOSIS IN THE NORTHWESTERN UNIVERSITY MEDICAL SCHOOL; EX-PRESIDENT CHICAGO NEUROLOGICAL SOCIETY.

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NERVOUS DISEASES.

SYMPTOMATOLOGY.

The Bechterew-Mendel Flexor Toe-reflex is made the subject of investigation by M. P. Nikitin, an assistant in Prof. Bechterew's clinic at St. Petersburg.[1] This reflex was first reported by Bechterew in February, 1901, as a special "tarsophalangeal reflex," called forth by striking the dorsal side of the tarsal bones or the bases of the metatarsal bones, and consisting of a slight flexion of the toes. Three years later Kurt Mendel published a report in which he stated that in a few cases of organic disease of the nervous system presenting spastic paralysis, striking the outer portion of the dorsal side of the foot "near the proximal portion" would cause a plantar flexion of the toes, with the exception of the great toe. The reflex, he said, was best obtained by striking about the center of the third and fourth metatarsal bones, or the cuboid or external cuneiform. If, on the other hand, the case was not one of spastic paralysis, or was functional, or if the patient were healthy, the toes were always extended (flexed dorsally) upon such a procedure. In the majority of cases when Mendel found this reflex, Babinski's toe phenomenon was also present. Mendel proposed his reflex (1) to distinguish organic from functional lesions, and (2) to determine the paralyzed side in comatose states.

Of late the Bechterew-Mendel reflex has been attracting more and more the attention of neurologists, and many articles upon it have appeared. In the literature the reflex is best known as the Bechterew-Mendel reflex, but it also goes by the names of the tarsophalangeal reflex, the flexor toe-reflex, and the dorsal foot-reflex. [See Practical Medicine Series, 1908, Vol. X, p. 8.]

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(1) Berliner klin. Woch., Sept. 7, 1908.

Nikitin has carefully studied this reflex in 35 cases of spastic paralysis, comparing it with the Babinski toe-phenomenon. The results are here tabulated:

Babinski Reflex.	Bechterew-Mendel Reflex.	Spastic Paraparesis.	Hemiplegia.	Totals.
+	+	17 cases	3 cases	20 cases
+	—	7 cases	2 cases	9 cases
—	+	2 cases	2 cases	4 cases
—	—	0 cases	2 cases	2 cases

Thus in two cases each of spastic paralysis and of hemiplegia the reflex was present when the toe-phenomenon of Babinski was absent.

A Contralateral Plantar Reflex is mentioned by H. Steinberg,[1] an assistant in E. Stadelmann's clinic. In one of Babinski's own cases, a woman with a spastic hemiplegia of several years' standing, he observed that while irritation of the sole of the paralyzed foot caused the toe-sign to appear, strangely enough, irritation of the sole of the *sound* foot also caused the toe-phenomenon in the paralyzed member. This observation seems to have been forgotten until record of it was found by Steinberg when he searched the literature for this phenomenon which he had himself just observed. The French observers of late are giving attention to this contralateral reflex, which they call the "contralateral heterogeneous plantar reflex." Steinberg reports six cases, the last one being very interesting. In this case there was an early right-sided paralysis, which had disappeared except for a slight paresis, and a very severe recent left-sided paralysis. Stroking the right sole caused plantar flexion of both the right and left great toes; stroking the left sole gave extension of the great toe on the left side, and flexion on the right.

Steinberg believes the contralateral reflex to be of diagnostic value, occurring even when the Babinski reflex is absent. It indicates to him involvement of the cerebral part of the upper motor neuron, when it is found. Still Steinberg admits that more cases must be examined before this conclusion can be ardently maintained.

Modifications of Sensibility in Disease. T. A. Williams[2]

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(1) Berliner klin. Woch., Dec. 7, 1908.
(2) Amer. Jour. Med. Sciences, April, 1909.

calls attention to Head's later work upon the subject in an article upon the importance of modifications of the sensibility in the diagnosis of disease. According to Head,[1] there are not, as was formerly taught, impulses of touch, temperature, and pain, but instead impulses (1) of *deep* sensibility; (2) of *epicritic* sensibility, and (3) of *protopathic* sensibility. (1) The fibers of *deep* sensibility originate in the muscle spindles and analogous bodies, and accompany motor nerves, and those in tendon, joint, and connective tissue in general. They are expressed in terms of pressure and attitude, and are commonly known as the nerves conducting muscle- and joint-sense impulses. They are also susceptible to vibration,[2] and when powerfully stimulated transmit impulses felt as pain. When they are diseased, ataxia and astereognosis follow. (2) *Epicritic* impulses proceed along the cutaneous nerves and consist of impulses of (a) light touch, (b) fine distinctions of coolness and warmth, and (c) the appreciation of two or more spots touched simultaneously, along with localization. (3) *Protopathic* sensibility enables us to perceive only the extremes of heat and cold, and only vaguely the tactile and painful impressions which are badly localized. Head believes that it is this form of sensation which is stimulated in visceral disease, and he lays stress upon the discomfort of an inexplicable kind which often accompanies the stimulation of these fibers. Of all our sensations, this form is the least differentiated phylogenetically. Head thinks it is probably that part of the afferent arc which expresses itself efferently in the pilomotor, vasomotor, and secretomotor reflexes. Unlike the epicritic system, the protopathic has no end-organs for the perception of heat and cold, and it is this system which shows the great overlapping discovered and investigated by Sherrington.

The groupings of the fibers carrying impulses of deep sensibility, of epicritic and of protopathic sensibility in the spinal cord are much involved. The painful impulses are conducted along fibers in Gower's tract on the opposite side of the cord, very closely associated with fibers carrying the impulses of heat and cold. The impulses of deep

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(1) See Practical Medicine Series, 1906, Vol. X, p. 108.
(2) See articles on pallesthesia, Practical Medicine Series, 1904, Vol. X, p. 110.

sensibility travel along the fibers of the posterior columns on the same side of the spinal cord, those of which we are conscious passing to the brain through the columns of Goll and Burdach, and those of which we are not conscious passing to the cerebellum through the homolateral direct cerebellar tract or the heterolateral anterior spinocerebellar tract and in the *tractus spinothalamicus et tectalis.* The impulses of epicritic sensibility also take two courses, some proceeding upward through the homolateral posterior columns for a few segments only, with the impulses of muscle- and joint-sense, and others proceeding upward through the heterolateral anterior column.

"Methods of investigation in practice: (1) Epicritic sense: (a) By drawing soft cotton-wool over the skin; (b) by localization; (c) by a pair of compasses; (d) by test-tubes between 22° C. and 47° C. (2) Protopathic sense: (a) By test-tubes nearer the freezing and the boiling points; (b) by the prick of a pin; (c) by an iron-cored faradic current. (3) Deep sensibility: (a) By pressure on the part; (b) by altering the attitude of a limb and asking the patient to describe or imitate the new position; (c) by the estimation of weights (muscular sense); (d) by the estimation of deep pain with the algometer. The tuning fork affects all these modes, and they are all necessary in stereognosis. (4) Cerebellar sense: (a) By the chair-mounting test; (b) by the diadocokinesis test; (c) by the revolving platform; (d) by the static equilibrium test.

"Experience teaches the neurologist to view with the greatest scepticism all evidence depending upon the statement of the patient, knowing empirically how they are fallacious even when without intent to deceive; and knowing psychologically that this must be so, on account of the very defective power of observation and introspection of the untrained laity. Some of the traps devised are: (1) La piège du peaucier of Jacquet; (2) Mankopf's pulse-sign; (2a) pupil-dilation sign; (3) simultaneous pressure trap; (4) faradic current trap; (5) counting touches; (6) diversion of attention."

Cutaneous Hyperalgesia. The diagnostic value of cutaneous hyperalgesia in abdominal disease has been recently investigated afresh by C. A. Elsberg and H. Neuhof.[1] Too little attention has thus far been paid to Henry Head's investigations and discoveries in this field, the literature being rather meager. The American authors first describe Head's zones and then enumerate their methods for testing cutaneous sensitiveness. They then proceed to the account of their study of the appearance and disappearance of zones of hyperalgesia in abdominal disease, the general significance of localized areas of cutaneous hyperalgesia in visceral disease, and the hyperalgesic zones in both the trunk and head in visceral disease. Many clinical cases to illustrate the conclusions arrived at are inserted. These conclusions follow:

"(1) We agree in the main with the statements of Head, that there are present, in many affections of the abdominal viscera, constant and definite areas or zones of cutaneous hyperalgesia.

"(2) These zones may vary somewhat in extent and outline, but they have a characteristic location.

"(3) The presence of a characteristic zone is an evidence of an affection of the corresponding abdominal viscus, although not of necessity the affection which is causing the symptoms.

"(4) The zones are present in a large percentage of patients with acute affections of the appendix, of the gall-bladder, of the uterine adnexa, and are of considerable value in the diagnosis of these acute affections.

"(5) Zones are frequently present in acute diseases of the other abdominal viscera, and when present aid in making the diagnosis.

"(6) Cutaneous hyperalgesia may appear early in acute abdominal disease. Its presence is no index of the gravity of the lesion. Its sudden disappearance may be of grave significance."

For the convenience of those not having access to Head's original papers, it may be stated that the important visceral zones are as follows:

Stomach: sixth, seventh, eighth, and ninth dorsal.
  Cardiac end: sixth and seventh dorsal, right.
  Pyloric end: eighth and ninth dorsal, left.

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(1) Amer. Jour. Med. Sciences, November, 1908.

Liver: eighth, ninth, and tenth dorsal, right.
Gall-bladder: eighth and ninth dorsal, right.
Intestines: ninth, tenth, eleventh, and twelfth dorsal.
Colon: ninth, tenth, and eleventh dorsal.
Cecum and vermiform appendix: tenth and eleventh dorsal, right.
Kidney: tenth dorsal, sometimes eleventh dorsal.
Ureter: eleventh and twelfth dorsal, first lumbar.
Bladder: (first?), second, third, and fourth sacral.
Uterus: tenth, eleventh, and twelfth dorsal, first lumbar.
Appendages: eleventh and twelfth dorsal, first lumbar.

Apraxia. Apraxia has been defined, says E. Régis,[1] as a motor disturbance independent of either paralysis or agnosia, consisting in the impossibility of executing acts adequate for the execution of a purpose, and seems to depend for its existence upon the obliteration of the motor images necessary for the performance of an act. At present two well-defined types of apraxia exist, the ideomotor apraxia or simple motor apraxia of Liepmann, and the ideational apraxia of Pick. In the former the apraxia is habitually partial, localized upon one side of the body, or occurring with varying intensity in different members. It is manifested in simple, elementary and imitated acts, and gives place to meaningless, amorphous movements. It is usually a transitory symptom, sometimes a residual phenomenon. Pick's ideational apraxia depends upon a lack of order in the partial acts necessary for the execution of the main purpose, but the motor kinesthetic memories are conserved. This sort of apraxia is generalized and involves all segments of the body. It appears to best advantage in complicated acts. The elements of the complicated acts may be performed with precision, and indicate purpose, but they are interrupted, unfinished, merely approximate. They may be repeated, giving the symptom called preservation, or there may be the substitution of wrong elements for the true ones. In a word, ideational apraxia consists essentially in a disturbance of the constitution of the idea which directs the act itself; while simple apraxia is the consequence of a disjunction between

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(1) Jour. de Méd. de Bordeaux, Feb. 7. 1909.

the conscious constituent psychic parts of the guiding idea, on the one hand, and their motor representation, on the other. In simple motor apraxia the members, so to speak, do not obey the will; in ideational apraxia the passive obedience of the members is perfect, but the orders received from the will are illogical and disordered.

Motor apraxia and ideational apraxia may occur separately and be almost completely independent of one another; but more frequently ideational apraxia is associated with motor apraxia, agnosia, confusion, and even paralysis. Ideational apraxia is necessarily very difficult to differentiate from agnosia, not merely because the two disturbances frequently coexist, but also because they are relatives. The inability to recognize an object, in other words, would be expected to be associated with an inability to recognize the uses of that object.

Régis insists upon one point rather strongly, that apraxia is a disturbance of the activity which is essentially voluntary, and is in no way related to automatic activity. Thus voluntary and automatic activities may be dissociated in apraxia.

G. Bromard and Miss Pascal[1] discuss the symptomatic value of apraxia, which they define as a lack of capability of realizing a movement in conformity with a given purpose, motility being preserved, however, and being in no way responsible for the incapacity. The apraxic understands, his limbs are not paralyzed, but he cannot execute, and consequently misuses the things which are given to him.

A given movement is usually executed in some such sequence as the following: (a) Fixation of the principal or final general idea, and its splitting up into secondary or intermediate ideas. (b) Transformation of these secondary or intermediate ideas into component motor images. (c) Transformation of these motor images into motor innervations. If, therefore, any one of these three operations is interfered with, apraxia results, and there may be, therefore, three kinds, as follows: (1) In the first case the main purpose is at fault; there is no general, guiding idea. It is not a case of faulty obedience of the members to the will, but rather it is the case of the will delivering faulty orders to the members. This is Pick's ideational apraxia.

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(1) La Presse Méd., April 17, 1909.

(2) In the second type the trouble lies in the splitting up of the main idea into the component parts, due to a break in the connections between the central psychic center where the plan of the act originates, and the secondary projection centers. The members obey the will badly in this case because of this dissociation between the kinetic formula orderly constructed, and the secondary kinesthetic images. In other words, the patient is incapable of transforming into motor images the plan of the act which was primarily correctly conceived. This has been called ideomotor apraxia by Liepmann; and better, transcortical apraxia by Heilbronner.

(3) In the third type the disturbance lies in the motor innervation stage, due to an alteration in the secondary projection centers themselves. The members do not obey the will because the innervation processes are incorrect. This is called simply motor apraxia, and by Heilbronner cortical apraxia.

Symptoms. Clinically, apraxia of the ideomotor type manifests itself in three ways: (1) by substituted movements (parapraxia, corresponding to paraphasia where words are substituted); (2) amorphous movements (jargonapraxia, corresponding to jargonaphasia; (3) and by absence of movements (apraxia proper, corresponding to motor aphasia).

Examination of the patient. The following table best illustrates the method of examination to be adopted:

"1. Autokinetic movements: walking, rising, sitting, dressing, undressing, etc.

"2. Simple movements: Close the eyes. Open the mouth. Put out the tongue. Cross the arms. Raise the right arm. Raise the left arm. Hold out the right hand. Hold out the left hand. Spread the fingers. Cross the legs. Raise the right leg. Raise the left leg. Put the foot forward, etc.

"3. Movements demanding thought: Point to the right eye. Point to the left ear. Pull the beard. Scratch the head.

"4. Expressive movements: Throw a kiss. Put your thumb to your nose. Give the military salute. Make the sign of the cross, etc.

"5. Descriptive movements: Pretend playing on a piano. Pretend turning a hand-organ. Pretend grinding coffee. Pretend to catch a fly, etc.

"6. Complicated movements: Fill a glass with water. Light a candle. Tie a knot. Seal an envelope, etc."

Diagnosis. It is first necessary to eliminate word-deafness and agnosia. In the former the commands are not heard because words are not heard as words; and in the latter, the words, though heard, are not perceived. It is also necessary to rule out ataxia, chorea, athetosis, etc., since faulty movements may result from these causes instead of apraxia. Given the diagnosis of apraxia, it is next necessary to diagnosticate the variety.

"As a general principle one might say that ideational varieties of apraxia are symptomatic of diffuse lesions, and that motor varieties are symptomatic of circumscribed lesions. In ideational apraxia there can of necessity be no question of localization. In the other two varieties of apraxia the apraxia is manifested on the side opposite the lesion."

Stereoagnosis and Tactile Asymbolia are ably and extensively discussed by Felix Rose and Max Egger.[1] Stereoagnosis is the result of experience, as is proved by the fact that it does not develop in cases of infantile hemiplegia occurring at a very tender age; and that it can be developed in the feet of those born without hands. This experience is stored-up memory images, according to Wernicke, of various sorts of sensory impressions. Sensory impulses excite two centers: there is the primary identification center of Wernicke, where we have the phenomenon of primary recognition; and a secondary identification center, where the name of the object is called forth from the sum total of sensory images which are the result of association. Disturbance of the former center constitutes stereoagnosis; of the latter, asymbolia. Both stereoagnosis and asymbolia are varieties of agnosia. A third variety of agnosia exists. In this variety the sensations are intact, the memory-

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(1) La Semaine Méd., Oct. 28, 1908.

images preserved, but the synthesis of the sensations does not take place, and the image is not called forth. For example, a patient of Wilbrand's, who could call to mind visually the streets of Hamburg, got lost among them when he ventured out; and a patient of Lissauer's, who could from memory tell the form and size of objects, did not recognize them when he saw them. This variety of agnosia is difficult to distinguish from asymbolia.

When one runs over the numerous observations of stereoagnosis in the literature, one is struck by the fact that simple cutaneous sensations are intact, while the muscle-joint sense is particularly apt to be affected. In other words, the superficial sensory impressions are not at fault, but rather the deep sensory impressions. Possibly the sensations of temperature and of pain may be associated in an accessory manner with stereoagnosis. Indeed, according to Rose and Egger, the word stereoagnosis should be reserved for those cases with cerebral lesions, in which touch is intact, so that in spite of an intact cutaneous sensibility there remains an impossibility of recognizing the form, consistence, surface, etc., of an object. It is in the recognition of these qualities that primary identification resides.

Has stereoagnosis a localization? Observations by many, the cases in point being reviewed, show that such a center lies in the superior parietal convolution. Among others, the case of Walton and Paul[1] is cited. (See Practical Medicine Series, 1902, Vol. X, p. 107.) Moreover, the authors themselves report at length the case of a man 33 years of age, in Raymond's service at Salpêtrière, who could not recognize objects, though he could tell their principal characters as to form, consistency and composition. They say: "After numerous controversies, there is a tendency to admit, especially under the impulse given to the subject by American authors [who are very freely referred to in this article.—Ed.], that the field of projection of the general sensory pathways is the superior parietal gyrus, to the exclusion of the ascending frontal. It is behind the ascending parietal, in the parietal gyri proper, that Wernicke, Mills, and Weisenburg seek to localize muscular

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(1) Jour. of Nerv. and Ment. Dis., April, 1909.

sensibility and the stereognostic perception, and Verger likewise believes in the localizing value of stereoagnosis." [See Verger's article, Practical Medicine Series, 1903, Vol. X, p. 146.]

The authors' conclusions follow: "(1) The term stereoagnosis should not be made use of in cases of gross sensory disturbances. (2) At the present time there exists no pure case of primary tactile agnosia except in those cases where the recognition of form never developed, as in cases of infantile hemiplegia coming on at a very tender age. (3) There are cases of pure tactile asymbolia. (4) Even if it is possible that a deficit in information by reason of subcortical hyperesthesia may lead to the syndrome of primary agnosia and tactile asymbolia, something as yet not proved by a single autopsy, the presence of these syndromes with slight sensory disturbances is in general favorable to the diagnosis of a cortical lesion. (5) It is in the course of chronic anatomical processes, and of microscopic processes, that there is occasion for meeting in a pure state, primary agnosia and tactile asymbolia, which names, contrary to what Morton Prince says, deserve to be retained, not to designate theoretical ideas, but realities, at least as far as tactile asymbolia is concerned."

Cytodiagnosis, Massive Coagulation. Cerebrospinal fluid coagulating *en masse* as soon as drawn, showing also a yellowish color, is reported by Sicard and Descomps.[1] The patient, a woman 39 years of age, addicted to alcohol, but not a syphilitic, was admitted to the hospital with a paraplegia of painless development, which in a few months placed her irrevocably upon her bed. She had bilateral contractures, disturbances of her sphincters, trophic ulcers, and marked sensory disturbances, a diagnosis of meningomyelitis of subacute and progressive type, being made. Four lumbar punctures were made. In the first and third a yellowish fluid issued which at once coagulated *en masse.* After standing a few hours a yellowish liquid issued from a fibrinous reticulum. After centrifuging, numerous lymphocytes and red blood-cells were found. The second and fourth punctures, oddly enough, gave a clear, limpid fluid, not coagulating, but containing a number of lymphocytes.

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(1) Gaz. des Hôp., Oct. 20, 1908.

At the postmortem examination a grayish white mass was found invading the epidural space at the level of the eleventh and twelfth vertebræ, at places adherent to the dura mater. The thickened mass encircled and compressed the roots of the cauda equina. Histologic examination of the mass showed numerous giant-cells. The process was undoubtedly bacterial, but the tubercle bacilli could not be found in the tissue. Inoculation experiments were not made, so that some doubt as to its etiology remains.

No case could better illustrate the morbid humoral type of cerebrospinal fluid, since all three of its characteristics were present: (1) the coagulation *en masse;* (2) the xanthochromia, and (3) the hemolymphocytosis. It is to be correlated with the other cases reported by Froin, Babinski, Cestan and Ravaut, and others. In the first part of Froin's cases there was a diffuse cerebrospinal meningitis and in the other two cases paraplegia of the inferior extremities. Babinski's case was one of such paraplegia, and so was the case of Cestan and Ravaut.

Sicard and Descomps, in trying to explain the pathogenesis of the fluid and its irregular occurrence in their case, affirm the existence of a circumscribed process of inflammation causing compression of blood-vessels and a consequent local edema. Thus is created a meningitic pocket into which the diapedesis of the red and white blood corpuscles occurs, and an exudation of blood-plasma.

These occurrences may be of practical value in diagnosis. Given a case of paraplegia of doubtful origin, with a question as to a meningomyelitis or compression of the cord by a tumor, the finding of a cerebrospinal fluid like that in this case would at once decide the cause to be a meningomyelitis. Thus it might have a decided bearing in deciding the question as to operative interference or not.

A second case of massive coagulation of cerebrospinal fluid in the course of meningitis is reported by G. Froin and G. Foy.[1] The case is further important as indicating grave danger in the use of collargol in the spinal canal in certain cases. The patient, twelve days after an operation for hydrocele, done after an injection of stovain in the spinal canal, developed signs of cerebrospinal meningitis and a turbid fluid on lumbar puncture, containing many polymorphonuclear cells. No bacteria were found, and cultures of bouillon remained sterile. After several remissions and recrudescences of the symptoms, on the twenty-sixth day of the malady there was found, after another increase in the severity of the symptoms, a complete transformation of the cerebrospinal fluid. It had become yellowish green, and coagulated *en masse* in fifteen minutes' time. Albumin was very abundant, alexine was found present on testing; and it gave the reaction for bilirubin when Gmelin's test was applied. A large number of red cells were found in it, 2,300 per cubic millimeter, and 1,480 white cells. Subsequent lumbar punctures did not give the same fluid. This changed condition of the cerebrospinal fluid followed the injection of collargol made on the tenth day of the illness, but there was probably no causal relation between the two facts. On this same twenty-sixth day, immediately following the withdrawal of this unusual cerebrospinal fluid, another 5 cubic centimeters of collargol was injected. This last injection, it was found the next day on lumbar puncture, was not absorbed, particles of collargol being found both within and without the leucocytes. On this occasion, though the fluid did not coagulate, red blood corpuscles were found in large numbers (40,000 per cubic millimeter), and 494,000 white cells were counted to a cubic millimeter. After this the patient became semicomatose and on the thirtieth day was removed by his friends from the hospital.

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(1) Gaz. des Hôp., Nov. 19, 1908.

Previous to this time the authors had made numerous injections of collargol into the spinal canal without any untoward results. In the present instance it was unsuccessful because of an unusual condition in the spinal canal. This was very likely a growing together of the cord membranes in such a way as to cause vascular compression and consequent local edema, the authors accepting the explanation of Sicard and Descomps. There is probably a modification of the arachno-pial cavity, consisting in the formation of pockets. Into these pockets, by a lowering of pressure, there pass the fibrinous serum and the cells, the decomposition of the red cells causing the yellowish color and giving bilirubin as a derivative of hemoglobin, thus rendering Gmelin's test positive.

NEUROSES.

HYSTERIA.

[During the past year there has been going on in France a very active discussion as to the nature of hysteria, and two representative articles upon the subject are here reviewed.—ED.]

The Nature of Hysteria forms the basis of a long paper by Babinski,[1] who defends his term *pithiatism* and pleads for its general adoption. Babinski,[2] brought up as he was by the teaching of Charcot, whose chief of clinic he was from 1885 to 1887, believed thoroughly in the old concept of hysteria until his observations led him in 1901 to make a communication before the Paris Neurological Society, in which he insisted that much was placed in the symptomatology of hysteria which did not truly belong to it. This new idea he has ever since elaborated, and especially during 1908, before the Paris Society of Neurology, has energetically and, it may be added, convincingly maintained. Among others, Brissaud, Dupré, Ballet, Souques, and Meige share his opinions more or less completely. The reason for the excessive extension of the symptomatology of hysteria is due to three leading causes: (1) errors of diagnosis, many organic cases being considered hysterical; (2) failure to detect simulation; (3) failure to distinguish various nervous states which ought to be distinguished one from another.

(1) Errors of diagnosis, in which organic lesions have been ascribed to hysteria, are exceedingly numerous. Take, for example, hemiplegia. Numerous are the organic cases diagnosticated as hysterical, in the days when physicians

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(1) La Semaine Méd., Jan. 6, 1909.

(2) Babinski's article on the dismemberment of traditional hysteria, and pithiatism, published in La Semaine Médicale, as the first paper in 1909, was translated in full by Charles Gilbert Chaddock, and was published in the Interstate Medical Journal, March, 1909.

had not the signs for their differentiation which we now possess. How could such errors have failed to arise? And our concept of hysteria was largely based on supposed facts gathered at a time when there were no differential signs of value known to the examiner. Now that there are many such differential signs hysterical hemiplegia is not found so frequently, and when it is found, it is found without any alteration whatever of the deep reflexes, these being never altered in hysteria. Thus, much has been swept from the symptomatology.

(2) Simulation is unceasingly found in the history of hysteria, resulting as a rule from a marked desire for notice or for sympathy. Sometimes the patient is, as it were, only a semi-simulator, not being conscious of the real mental source of his complaints and deeds. Much of the simulation is actually suggested to the patient by the examining physician, as the limitation of the field of vision and various sensory disturbances. Other symptoms complained of are simply due to rank deception or fraud, being beyond the pale of auto- or hetero-suggestion. Thus erythemata, ecchymoses, ulcerations, sores, edemata may all be made to appear on the surface of the body. These are real enough in themselves, but their origin lies in deception and fraud.

(3) Lastly there are numerous neuroses or nervous states, not hysteria, which have been ascribed to it.

On looking over the whole symptomatology of hysteria as it at present appears, it will at once be apparent that the symptoms split into two great groups, (1) those which can be produced by suggestion and (2) those which cannot be so produced. Babinski insists that only the former should be regarded as hysterical symptoms, and not one of the latter. Babinski, however, uses the word suggestion in a technical manner, rather than in its own strict meaning. According to him a symptom is to be regarded as due to suggestion only when the will is complete mistress of the symptom, being therefore able to cure it. Thus the word suggestion by definition contains two elements, implying first that the will is capable of producing a symptom, and secondly, that it is capable of removing it when produced. Should either of the elements be lacking, the symptom is not truly hysterical. Thus symptoms which may be produced by suggestion, and are therefore hysterical, are: convulsive crises, paralyses, contractures of every sort and location, various tremors, choreic movements, disturbances of phonation, and of respiration, sensory disturbances such as anesthesia and hyperesthesia. Symptoms not capable of being produced by suggestion and therefore not hysterical are: exaggeration or loss of the tendon-reflexes, disturbances of the pupillary and cutaneous reflexes, vasomotor, secretory and trophic changes, hemorrhage, anuria, albuminuria and fever. Concerning these two groups of symptoms, there seems rather complete agreement with Babinski on the part of his fellow members in the Paris Neurological Society. To the former group of symptoms Babinski has applied the term pithiatic, and to hysteria with a symptomatology limited to such symptoms only as can be made and removed by suggestion, he applies the term *pithiatism,* from the Greek, meaning *to persuade* and *curable.*

Hysteria has often been ascribed to emotion, but in Babinski's opinion emotion seems to have little to do with its production, far less, for example, than has imitation. It is not so much emotion, as a desire to arouse emotion in the circle about, and often it is merely ordinary simulation. Marinesco[1] while reporting a case of aphasia in hysteria, refers to Babinski's views about emotion as an etiological factor (see page 27) and disagrees with them, ascribing the outbreak in the case he reports as due to fear. Babinski's concluding paragraphs follow:

"The ideas which I have just expressed seem to me worthy of consideration, not only because, being true, they deserve in common with all scientific truths, to be dwelled upon; but also because many practical consequences flow from them which I shall successively indicate.

"(1) Being aware of the effect which he exerts upon impressionable subjects and the rôle which he must involuntarily play, the physician should observe himself quite as much as his patient; he should weigh all his words, bearing in mind that a badly worded question or an inopportune remark may lead to some suggestion. This danger he must never lose sight of, and he will avoid it if he limits himself in his questions and examinations to those rules which I have elsewhere indicated, in their general outlines.

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(1) La Semaine Méd., June 30, 1909.

"(2) Knowing that a suggestionable subject readily yields to surrounding influences, he will not content himself with merely acting in person by his psychotherapeutic measures upon the patients affected with pithiatic disturbances; but he will use his ingenuity to create in every way possible a psychic medium which shall be salutary for them.

"(3) Convinced that pithiatic disturbances ought to yield quickly to a skilfully applied psychotherapy, the physician, when confronted with the failure of his therapeutic attempts in a case of this sort, will be led to conclude that success has been thwarted by some contrary mental suggestion; he will therefore seek to discover it in order to remove it, and thus bring about conditions which will assure a cure.

"(4) Knowing the demarcation of pithiatism he will know how to distinguish extraneous symptoms, and will not attempt to cure them by psychotherapy. Thus by abstaining from promises which he cannot fulfill, he will not bring discredit upon himself. Besides, and this is more important, having no illusions concerning the effects to be expected from such treatment, he will be less likely to neglect the therapeutic measures which might lead to a cure of the symptoms which are not pithiatic.

"(5) Having ideas concerning pithiatism and simulation which are more exact and precise than those formerly held, he will be more fit, and also more expert, in giving advice relative to damage suits in occupation accidents.

"(a) On the one hand, he will not make the mistake of attributing to hysteria symptoms which the will is incapable of producing, and which therefore are not pithiatic. (b) On the other hand he will be more able than ever before to oppose the abuses which the laws concerning accidents have engendered, which abuses the old conception of hysteria is one of the principal causes for the existence of.

"Permeated with the idea that 'the vasomotor and trophic hysterical disturbances' are merely fictions, that the conception of these pretended hysterical symptoms is based upon errors of diagnosis and exaggeration of one's condition, the expert will the better direct his researches, when he meets with a case caused by some accident, presenting, for example, visceral hemorrhages, ulcerations, gangrene, cutaneous hemorrhages, or edemata.

"I believe myself justified in concluding from this study that the old idea of hysteria based upon observations in part insufficient, in part erroneous, cannot stand up before critical examination; that the dismemberment of our old traditional hysteria is an inevitable consequence of a series of facts formerly ignored and solidly established to-day; that from this dismemberment results the liberation of an autonomous group of symptoms occupying a very important place in pathology, for which may be reserved the old denomination hysteria, but for which the new word pithiatism is a much more expressive name."

Present Status of Hysteria. An able article on the present status of hysteria by Tom A. Williams[1] is illuminating, being a careful review of the extensive discussion of the subject by the Paris Neurological Society during the past year. To systematize the discussion of hysteria, it was considered under eight different heads, which were later taken up separately, in order, placed in the form of questions, and voted upon by the members present.

"(1) The first question was: 'Among the phenomena usually included in hysteria, is there a special group of symptoms which can be exactly reproduced by suggestion and which can disappear under the influence solely of suggestion or persuasion? In particular, certain kinds of convulsive fits, paralyses, contractures, anesthesiæ, hyperesthesiæ; of modifications of the special senses and of difficulties of speech; as well as certain respiratory, digestive, and other troubles?' This question had been discussed in the Paris Neurological Society, principally at the instigation of Babinski, during the past six years. Hence little further talk was required before the question was answered unanimously in the affirmative. Even Raymond, whose ideas concerning hysteria are those which have been characteristic of the Salpêtrière since the days of Charcot, acceded, letting it be clearly understood, however, that he spoke of certain cases and not of all.

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(2) New York Medical Journal, Jan. 9, 1909.

"(2) The second question was as follows: 'If one admits the existence of a group of characteristic symptoms, is it not necessary to give these symptoms a name? The term of pithiatism having been proposed, should it be kept?' The second question was postponed to the end of the discussion, as it concerned terminology.

"(3) The third question concerned the genesis of hysterical symptoms; and it is of the greatest practical importance. It was as follows: '(a) Are not the so-called hysterical stigmata (sensitivosensorial hemianesthesia, contraction of the visual field, monocular polyopia, dyschromatopsia, loss of pharyngeal reflex, hysterogenetic zones, etc.) the result of an unconscious suggestion, usually of medical origin? (b) Is it not possible to admit that sometimes suggestion plays no part in originating the stigmata, and that they may develop spontaneously under an unknown influence?'

"Both Raymond and Déjerine firmly believe that cases occur where undoubted hysterical symptoms arise without any possible suggestive influence. They, however, are speaking here merely of direct medical or other suggestion. They do not consider the possibility of a suggestion induced by their own examination of the patient. Babinski once more insisted upon his method of avoiding suggestion in examining the sensibility, and both Ballet and Souques declare that nowadays, taking these precautions, they never find the stigmata in hysterics who have not been previously medically examined.

"Raymond, however, believes that this susceptibility to anesthesia is something special to hysteria. It seems very evident that the special predilection is one towards suggestibility, this being the cause of the anesthesia, ascertainable in the vast majority of cases in proportion to the technical skill used for its elucidation. Another factor was remarked by Meige, who pointed out that the examiner usually pricks the left side of the patient first, because this was opposite the right hand holding the pin; and as Pitres had to confess, asks the patient 'Do you feel?'; this in itself contains a suggestion that he may not feel, besides being subject to the fallacy of the patient's miscomprehension in believing that the question concerns only pain, which need by no means be caused by every pin-prick, as may be verified on any normal person.

"As it is impossible to prove a universal negative, question (b) is necessarily insoluble, logically speaking, more especially as such symptoms are frequently consciously simulated, and that the difference between conscious and unconscious simulation is moral and not medical; each of them, however, is equally derivable from suggestion, autochthonous or not.

"The majority of the society decided then that one finds medical suggestion at the root of a vast majority of hysterical symptoms in proportion as one seeks it with more intelligence and care, and that it is of course impossible to deny it even in most of those cases where it is not elicited.

"(4) The fourth question is as follows: 'Have suggestion and persuasion any action upon (a) the tendon reflexes, (b) the skin reflexes, (c) the pupil reflexes, (d) the circulatory and trophic functions (dermographia, urticaria, edema, eruptions, hemorrhages, ulcers, gangrene), (e) the secretory functions (urine, sweat, saliva), (f) the temperature (fever)?'

"Everyone agrees that suggestion has no power over the tendon reflexes, as they are beyond voluntary control; but the skin reflexes are more or less modified by the will, and it is very probable that they react through the cortex. Hence, they may be amenable to suggestion. Of course, any of these reflexes can be more or less simulated or suppressed intentionally; but even then the reproduction is incomplete, often exaggerated or ill timed, and can be distinguished from the true reflex by a discriminating observer with a little finesse. Every one nowadays agrees that suggestion cannot modify the reflex of the pupils.

"No member could affirm that he had ever by suggestion provoked dermographia, urticaria, edema, ulcerations, etc., though both Pitres and Brissaud believed that they had in the past seen cases which *seemed like* hysterical swelling of the breast. It is necessary here to take account of the *mythomanic* character of so many suggestible patients. As to hysterical fever, none of the observations hitherto published are beyond criticism as regards the precautions taken to eliminate on the one hand deception, and on the other intercurrent disease. It is certain that no one has succeeded experimentally in producing fever by suggestion.

"(5) The fifth question was as follows: 'In admitting that neither suggestion nor persuasion have any influence upon the above named phenomena (see fourth question) does there not exist a connection between these phenomena and the symptoms noted in paragraph 1; and is the connection one of causality, of interdependence, of association, or of simple coincidence?

"Some members were under the impression that they had observed facts indicating causality between these phenomena, especially as regards the vasomotor, though most believed it was one of simple coincidence, and Babinski urged the importance of eliminating trickery.

"(6) Question six began in a discussion as to the meaning of the word suggestion. Déjerine pointed out that all the acts of our life are influenced by suggestion. Even the most cogent reasoning for an opinion only convinces through the hearer's faith in the superiority of the reasoner. Babinski believes that the term suggestion should always imply a *pejorative* idea, that is to say, correspond to our use of the word insinuation; and that to the notion of making another person accept an idea that is reasonable the name persuasion should be given. On the other hand daily life furnishes numerous examples of *meliorative* persuasion. There is in fact 'a fundamental distinction between suggestion and persuasion, and this consists of the subject's entire unconsciousness, the absence of realization of the manner in which a new idea has been imposed upon him during suggestion. Sometimes he is even ignorant of having received anything new. Persuasion on the other hand appeals to the individual's own power of reflection and this makes him aware of the whole process of reasoning by which he becomes convinced.

"Suggestibility is diminished and persuadability fostered by rational education; and during the progress of this, the same individual may exhibit both reactions according to the intellectual habit aroused by the various stimuli employed. The rational psychotherapy of hysteria includes a reëducative procedure of this type. For the purposes of the discussion, the members accepted the use of the word advanced by Babinski. No member was able to give an instance of any other condition than hysteria which was curable by suggestion alone; hence question six must be answered in the negative.

"Question 8 was whether we should change the name hysteria to one less misleading.

"The name of psychosis by imitation was proposed by Dufour as being better than either pithiatism or hysteria; and Ballet pointed out that the infantile mentality must be included if Babinski's ideas prevail, and believes that hysteria is less the trouble suggested than the psychic manifestation, probably emotional, which permits it, as in the case of a traumatic neurosis, which for him is not the result but the cause of the suggestibility. No decision was arrived at; and the nomenclature difficulty along with Question 7 (upon the relation of emotion to the phenomena under consideration) has been postponed until the end of the year. Its discussion will be considered and the whole question summed up about that time."

Hysterical Aphasia. Hysterical aphasia has been denied, but it undoubtedly occurs and Marinesco[1] reports a case very clearly and forcibly. Cases have been previously reported by Charcot, Lépine, Raymond, Janet, Bernheim, Guillain, and Courmont. Courmont thus classifies cases of hysterical aphasia:

1. Complete type, aphemia, agraphia, word-blindness and word-deafness (author's case).
2. Incomplete and dissociate type, especially hysterical aphemia (type described by Charcot under the name of mutism).

Besides these two forms, there is a third which is not true aphasia but a voluntary mutism; in hysterical aphasia the patient *can* not speak, whereas in this form he *will* not speak.

A recent case of hysterical aphasia was shown at the Paris Neurological Society by Laignel-Lavastine and Glénard,[2] which does not belong to Courmont's types. The patient, a man with a right-sided hemiplegia and pure motor aphasia, both being hysterical, could only say: "a

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(1) La Semaine Méd., June 30, 1909.
(2) Rev. Neur., Dec. 30, 1908.

bibi," with various intonations and gestures, depending upon the meaning he sought to convey. In fact he made himself understood perfectly by a very expressive pantomime, in the practice of which he might almost have been called voluble, for he was always gesticulating. When gestures failed, he would write with his left hand very rapidly, without mirror-writing. His aphasia was purely motor. Tested by the Lichtheim-Déjerine test he would indicate the exact number of syllables contained in the word representing the object shown to him. There was no suspicion of word-deafness or word-blindness. Lumbar puncture showed a normal cerebrospinal fluid. The cutaneous and tendinous reflexes were normal; there was no ankle-clonus; Babinski's toe-sign was absent; Hoover's sign of complemental opposition was as it is in the normal individual, no hypotonicity was present, and in short there was nothing which stamped the case as organic. Moreover suggestion worked a miracle, the man being able after a few minutes' application of electricity to move his fingers, and then to walk. The mutism remained while people were about, but disappeared when he was alone with a single person.

Marinesco's case[1] was that of a midwife who, on being summoned to the bedside of a patient of hers who had just died, fell unconscious, apparently by reason of a stroke of apoplexy, for when she was examined later she showed a complete right-sided hemiplegia and a very peculiar speech disturbance, which the author called a paradoxic aphasia. She prolonged some of her vowels, omitted parts of words, using made-up abbreviations scarcely intelligible. There was no change in the reflexes, Babinski's toe-sign was not present, and no sign indicating an organic lesion could be discoverd. She was much depressed. Undoubtedly the cause of her affliction was the emotional shock occasioned by seeing one of her patients dead, notwithstanding Babinski's view that emotion is seldom a cause of hysteria. The patient was rapidly cured by persuasion, "psychologic conversations," in which the attempt was made to show her that her fright had not produced any organic effect upon her central nervous system.

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(1) Loc. cit.

The Reflexes in Hysteria have been studied anew by H. Roger.[1]

(A) The reflexes associated with the mucous membranes are only two, the pharyngeal and the corneal. The pharyngeal reflex, though often absent in hysteria, is very often present; and it is frequently absent in other conditions than hysteria, such as epilepsy, general paralysis of the insane, organic hemiplegia, lead poisoning, etc. It is even absent in people absolutely healthy. Its diagnostic value is therefore nil. The corneal reflex is often absent when the cornea is anesthetic, but not necessarily so. It is not abolished as a rule in hysteria; indeed it may even be exaggerated. It also is without diagnostic value.

(B) The pupillary reflexes are at times affected in hysteria. The reaction to light is almost never disturbed, but cases of hysteria are recorded of mydriasis with abolition of the light-reflex, especially by Westphall, who reports eight cases in his experience. The dilatation of the pupils on pain persists in hysteria even when the region in which pain is caused is anesthetic.

(C) Cutaneous reflexes. (a) Abdominal reflexes are variable in hysteria, sometimes being normal in hysterical anesthesia and sometimes abolished. As a rule they are about as often present as absent. (b) The cremasteric reflex is usually normal, but has been reported absent and also exaggerated. Even in hysterical hemiplegia it may be normal. (c) The plantar reflexes, of which there are three varieties: (1) the plantar reflex in flexion, (2) the reflex of the fascia lata, and (3) the defensive reflex in which the whole foot is jerked away, vary largely in hysteria. Abnormal responses, such as Babinski's toe-sign, the reflex of Schaefer (extension of the great toe upon pinching the skin over the tendo Achillis), Oppenheim's reflex (extension of the great toe on scratching the skin from above down in the lower part of the calf), and the reflex of Herschberg (adduction of the foot by slight irritation of the inner border of the foot at about the root of the great toe), are all absent in real hysteria. On the other hand the fan-sign (reflex abduction of the toes) is frequently present.

(D) Tendon-reflexes. (a) Exaggeration according to some occurs in hysteria, but not according to Babinski and his followers. Ankle-clonus has been reported, but really never occurs. A spurious ankle-clonus, less rapid, less regular, easily dying out, is on the contrary quite common. The Gordon reflex (extension of the great toe or all of the toes upon deep pressure of the calf muscles), and the dorsal foot-reflex of Mendel-Bechterew (flexion of the four lesser toes when one strikes with a percussion hammer the dorsal region of the foot over the cuboid and external cuneiform bones), are not present in hysteria. (b) Abolition of the reflexes is very rare in hysteria, if indeed it occurs at all. (c) Variability from day to day in the tendon-reflexes seems quite characteristic of hysteria.

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(1) Gaz. des Hôp., Dec. 1, 1908; Jan. 9, 1909.

Hysterical Monoplegia. A case of hysterical monoplegia consequent upon the appearance of a fibrosarcoma of the arm is reported by L. Rimbaud and J. Anglada.[1] The patient, a woman with a grown-up son, some twelve years before was paralyzed in her right arm in consequence of fright caused by a collapsing building. This paralysis yielded to electric treatment in three months' time. Shortly before the second monoplegia she noticed a little tumor on the arm which twelve years before had been paralyzed, and she was greatly concerned about it, fearing it was malignant, and therefore avoiding seeing a physician lest her arm should be amputated. Just at this time she again witnessed the fall of a château, and her son was brought home injured. She was immediately seized with a nervous crisis, became semi-conscious for a week, talking in a rambling way, and not knowing what she did. In a few days she quieted down, but again the right arm was paralyzed. Moreover sensation was lost, both to pain, temperature, and touch, involving the whole arm and stopping at a line exactly at the shoulder. The tumor looked like a fibroadenoma. The patient was of the opinion that the tumor was the cause of all her difficulty and she was encouraged in that idea.

So marked was the anesthesia that the tumor was removed without any anesthetic, general or local. The operation was absolutely painless, lasted four or five minutes, and was followed by thorough curettage of the adjacent

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(1) Gaz. des Hôp., June 17, 1909.

tissue. The first stitches were put in without being perceived, when the patient was told that the operation was nearly over. She immediately began to feel little crawling sensations in her arm, and groaned slightly when the last stitches were being taken. In three days the sensation was normal and the arm could be moved freely. After the fourth day the stitches were removed, and the progress became very rapid, so that within a week after the operation both sensation and motion were perfect.

Very interesting is the extent of the hysterical anesthesia which permitted the operation. There was no stoical endurance on the part of the patient; she simply did not feel. There was no simulation, for there was need of none. There was no pending law-suit, no settlement to be effected, nobody who could be blamed for her condition. Yet she had a severe monoplegia with anesthesia.

NEURASTHENIA.

At the tenth Congress of Medicine held during August, 1908, at Geneva, neurasthenia was one of the topics under special consideration, its *pathogenesis* being the central point of the discussion. Prof. Dubois of Berne insisted upon the psychic origin of neurasthenia, which he called a disease of the mind and not of the body, curable therefore only by psychotherapy. In this opinion he was supported by Déjerine. On the other hand Teissier, de Fleury, Deschamps and others insisted upon the physical origin of the disease, and to their views Bernheim in large measure inclined. Lépine[1] believes the divergence of opinion arose from the fact that different observers had not all seen the same patients. So extreme are the differences between one case and another that at the present time no one theory of pathogenesis can be demonstrated. There are assuredly two groups of cases: those cured by psychotherapy and those not so cured. The former are very "suggestible" individuals; the latter are not. It is the former group which Dubois has seen so much of, and to Lépine these cases appear more like psychasthenics than neurasthenics. In this opinion Bernheim also coincides.

Those not cured by psychotherapy will be found to

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(1) Lyon Méd., Oct. 4, 1908.

have about them an organic element which must be discovered and cured before the neurasthenia can be relieved. Therapeutic experience confirms this opinion. In some instances, it is true, an organic disturbance which subsequently disappears may usher in a neurasthenia which lasts indefinitely. In such cases the mental condition nurses the neurasthenic state though it does not create it.

Practically, therefore, concludes Lépine, the attending physician should be devoid of any tendency towards absolute acceptance of any one theory of pathogenesis, for the pathogenesis is undoubtedly very complex. When there is really an important and lasting organic cause, psychotherapy never will succeed. No one therapy can ever avail. Neither tonics nor isolation should be systematically used in all cases.

The *etiology* of neurasthenia is discussed by A. T. Schofield.[1] Neurasthenia is primarily due to heredity and malnutrition, while it is brought on by excessive strain or poison. Over-education is not the important factor it is supposed to be. In the eighteenth century this disease in England was called "the vapours," and was almost exclusively attributed to suppressed sexual passion. Schofield believes that to-day enforced celibacy amongst women is a much underestimated cause. Neurasthenia is also largely due to hereditary alcoholism, and to malnutrition and monotony of work. The leading causes of neurasthenia may be summed up as:—

1. *Poisons* from influenza, syphilis, enteritis, dyspepsia, alcohol, drugs, zymotic and other diseases.
2. *Malnutrition.*
3. *Fatigue* from overwork, worry, insomnia, pain, sexual excesses or suppression.
4. *Emotional strain* from shock, grief, accidents, religion, love, etc.
5. *Indirect causes,* as enteroptosis, floating kidney, eyestrain, also bad environment, suggestion, and other psychic factors.

The principal predisposing cause is heredity. Psychologically, neurasthenia is a disease of exhaustion of the higher nerve centers, since poisoning produces toxic effects

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(1) British Med. Jour., Sept. 19, 1908.

similar to those from overwork. To weakness of these centers by fatigue or poison from some of the above causes we may attribute even the most psychic symptoms of this disease, such as "phobias" and "obsession," provided always that the patient be free from any underlying mental taint.

EPILEPSY.

The Nature of Epilepsy. According to L. Marchand[1] epilepsy is not a disease, but a symptom, being comparable to dropsy, jaundice, etc. He examined the brains of intelligent epileptics without paralysis in life, and also the brains of epileptics with mental weakness, imbeciles or idiots. He arrived at the following results: Meningo-cortical adhesions more or less diffuse, but predominating over the frontal lobes and motor-areas, and slight superficial scleroses of the subjacent cerebral cortex are the only lesions met with in intelligent epileptics; the network of tangential fibers (the intellectual association fibers) is normal. In the brains of epileptics with imbecility or idiocy he observed in addition to the meningeal adhesions a more accentuated cerebral sclerosis with a disappearance of or a simple diminution of these tangential fibers, depending upon the intellectual level of the subject.

These lesions afford an explanation of the intellectual enfeeblement of a certain number of epileptics, and the complete imbecility, idiocy or dementia of others. The former group shows simply a symphysis between meninges and cortex with little or no effect on the cortex; the latter shows a clearly marked meningoencephalitis. Epilepsy ought, then, to be considered as a symptom of a brain disturbance rather than as a morbid entity. The author concludes as follows: (1) Epilepsy is a syndrome which appears the more frequently in the course of brain disease, the younger the patient. (2) In the child and the youth epilepsy is very frequent in all the diffuse maladies of the meninges and cortex, whether acute, subacute, or chronic. (3) In the adult and the aged epilepsy appears especially in meningo-cortical affections of acute or subacute type, being rare in chronic affections.

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(1) Gaz. des Hôp., Feb. 2, 1909.

Epilepsy in Secondary Syphilis. Epilepsy occurring during the secondary stage of syphilis, though very rare, has nevertheless been recorded; and L. Guénot[1] now records a case. Epileptic convulsions during the secondary symptoms usually manifest themselves as a partial epilepsy of the Jacksonian type, but they may be of the usual typical sort found in idiopathic epilepsy. The patient, a young man twenty-six years of age, acquired a chancre at the beginning of December, 1908. On Dec. 31 he had the first convulsion he had ever had in his life. A second followed Jan. 15, and a third Feb. 10. The first convulsion was the most severe, and resulted in a fall which made a large occipital scalp wound. There was no incontinence of urine except in the first convulsion. Feb. 21, nearly three months after getting the chancre, the patient showed a slight lymphocytosis in his cerebrospinal fluid from which the Wassermann reaction was obtained by Dr. Salmon at the Pasteur Institute. Vigorous treatment cleaned up all of his symptoms so that by the end of March he was able to leave the hospital. Just before he left, the Wassermann reaction applied to the cerebrospinal fluid gave a negative result.

The syphilitic nature of these convulsions is indicated by their coincidence with the other syphilitic secondary symptoms, and by the curative influence of the specific treatment. Symptomatically syphilitic secondary epilepsy [Fournier has recorded twelve cases] does not differ from idiopathic epilepsy, unless it be in the absence of the initial cry. The aura may be present and was present in the case here reported. The noteworthy point about this case is the early appearance of the convulsion (within one month), most cases not showing convulsions until at least six months after the acquisition of the chancre. The prognosis is fairly benign, far more so than the epilepsy of tertiary syphilis, in which the prognosis is poor. Another noteworthy fact in this case is the early appearance of the Wassermann reaction in the cerebrospinal fluid.

Senile Epilepsy, and the vertiginous attacks which supervene for the first time in advanced life, are discussed by T. D. Savill[2] and are illustrated by a case of cardio-

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(1) Gaz. des Hôp., June 10, 1909.
(2) The Lancet, July 17, 1909.

arterial hypermyotrophy. The patient, 62 years of age, had his first convulsion some 18 months previous to consulting Savill. The fits were severe, with all the characteristics of idiopathic epilepsy, beginning with a visual aura, and a feeling of heat in the face. Examination showed a blood-pressure of 210 mm., a pulse-rate of 96, and hypertrophy of the heart with some dilatation. The causes of senile convulsions are idiopathic epilepsy, hysteria, cerebral syphilis, cerebral tumor, chronic degenerations of the nervous system, cerebral pachymeningitis, uremia, diabetes, and various other toxic conditions, such as those caused by various drugs, lead poisoning, and chronic alcoholism. Other causes are *circulatory,* comprising hemorrhage, thrombosis, and embolism, cardiac weakness, and arterial disease, high blood-pressure, and cardiovascular hypertrophy of the myocardium. Arterial disease is the most frequent cause of syncopal and vertiginous attacks in the aged, even without any discoverable cardiac defect. The case herewith reported might be regarded by some as chronic Bright's disease, but all the symptoms presented were circulatory.

"By way of *summarizing the pathology* of the various forms of syncopal, vertiginous, and epileptiform seizures which occur for the first time in advanced life my experience among the aged has led me to the following conclusions:—

"1. That idiopathic epilepsy never arises for the first time in advanced life.

"2. That at least nine-tenths of these syncopal, vertiginous, and epileptiform seizures are circulatory in origin.

"3. That senile syncope is generally due to cardiac failure and low blood pressure.

"4. That senile vertigo and similar head sensations (postural vertigo, interruptions of thought, etc.) are due to the disturbances of the regulator mechanism of the arteries in different parts of the body owing to arterial hypermyotrophy or other arterial disease, not necessarily accompanied, as far as my observations go, by any notable alteration of the general blood pressure or of cardiac increase or diminution.

"5. That senile epilepsy (convulsive attacks) is generally due to increased blood pressure with cardio-arterial hypermyotrophy.

"All these attacks of senile syncope, senile vertigo, and senile epilepsy merge into and are associated one with the other; vertigo may occur at one time, convulsions at another, and the same patient may in the end die from syncope (when the heart is defeated)."

The Psychoses of Epilepsy and their *treatment* forms the subject of a paper of length by E. Siemerling.[1] According to his experience about one-fifth of all cases of epilepsy remain entirely free from psychic disturbances, but the other four-fifths suffer from them in varying degrees. He distinguishes chronic epileptic mental disturbances (epileptic dementia) and acute and transitory epileptic mental disturbances, which are called pre- or postepileptic or pre- or postparoxysmal disturbances, or the *equivalents,* it being a requirement that such disturbances occur independently of a convulsion.

Epileptic dementia or idiocy may reach an extreme degree. About half of all idiots are epileptics, and many of them show organic brain disease. Besides epileptic idiots there is a group of epileptics made up of those who undergo a gradual regression, showing increasing mental deterioration. This may take place rapidly at the time of puberty or may come on in association with arteriosclerosis in senile epilepsy or epilepsia tarda. There are evident in these cases a diminution of the moral and ethical sense, irritability, outbursts of emotion and even of brutal actions. Characteristic is the fact that the ethic defect antedates the intellectual one. Usually, too, these individuals are egoists, incapable of judgment, given to lies, mistrustful, unsteady in their work. Among tramps and prostitutes a not inconsiderable number belong to this group. Often these cases show an inclination towards the mystic, and towards religious fanaticism.

The acute and transitory disturbances or the *equivalents* resemble true epilepsy in that there is in them an alteration of the state of consciousness, ranging all the way from slight mental haziness to complete loss. The change of

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(1) Berliner klin. Woch., Jan. 4, 1909.

consciousness shows itself in delay and in difficulty of thinking, in incoherence or the dissociation of a chain of thought, in consequence of which there follow irregular, disordered actions, or a mixture of orderly acts and most unusual, unexpected acts of a violent nature. Siemerling classifies these cases thus:

1. Disturbances of the affectivities (moodiness, often sad moods, dysphoria, increasing to melancholia and mania).
2. Confusional mental states (stupor and delirium, usually with hallucinations, increasing to frenzy).
3. Paranoid states and paranoia.
4. Dream-states.

1. Disturbances of the affectivities often begin with complaints as to all sorts of subjective sensations, followed by hypochondriacal ideas, such as that the brain moves, the heart stands still, the body is hollow, the brain has holes in it, etc. Sometimes these hypochondriacal fixed ideas take on a paranoiac tinge, in that the victim makes his surroundings accountable for his fancied condition. In other cases melancholia follows, with self-reproachings, and suicidal tendencies. Often the patient complains of a strange, uncanny feeling; he thinks of something horrible or has an idea that something is impending. His feeling of tension may lead to a sudden impulsive act of a criminal nature. More rarely the mood is an unnaturally cheerful one, leading to mania-like states, or euphoria. In these conditions hallucinations are often encountered.

2. Epileptic confusional mental states, with stupor and delirium mixed up hopelessly in the same individual, are not and cannot be clear cut. Generally the stupor or the delirium predominates, but they often alternate with each other. In *stupor* the patient is apathetic, cannot be aroused, and if he does anything, does it like an automaton. Yet even in automatic actions there is a marked tendency toward sudden outbursts of phobias or of passion. Sometimes there is what may well be called katatonic stupor, sometimes a stuporous slumber-state (narcolepsy). In the *delirium* case, there is complete disorientation as to time, place, environment, and even one's own individuality. Sometimes the picture reminds one of alcoholic delirium. There are numerous hallucinations, and illusions. There may be a state of ecstasy or of transitory mania. Some-

times there are delusions of persecution, often of a religious nature, and these delusions may be of a paranoid type.

3. Paranoid states may be a part of a general disturbance of the affectivities as has already been noted, or of epileptic hallucinatory delirium. A special form, with a well organized system of ideas of persecution and of grandeur is known as *paranoia chronica epileptica.* This condition begins in an ordinary epileptic mistrust. The patient thinks his surroundings to be false; something is going on that he does not know about; he is being watched; the doctor and nurse make certain motions; the food is specially prepared. Hallucinations and illusions confirm his delusions. The pent-up fury suddenly bursts forth in a tempest of rage. Ideas of grandeur like those of ordinary paranoia occur. Often these are of a religious nature, God speaks to the patient, he sees God, hears heavenly music, etc.

4. Dreamy states or the dawn states of the French are often of short duration. The consciousness is beclouded, memory is impaired, everything has a veil thrown over it. In this state we find the double ego, the dual personality. The patient looks tired and dull, the eyelids droop, he starts as though frightened when spoken to, he repeatedly yawns, the speech is slow and difficult, suggestibility is increased, the gaze is fixed as in hypnosis, and he may look as though he were in a state of fearful oppression. Numerous vasomotor disturbances are associated. Often there is perseveration, verbigeration and echolalia. There is amnesic aphasia, and often articulatory disturbance of speech. In the motor sphere there is stereotypy, echopraxia, tremor, disturbance of equilibrium, and frequently chronic spasms (chorea, athetosis, myoclonia). These dream states may be combined with any of the three preceding groups in any proportion or mixture.

Treatment holds out hope of a measure of success. It should be both prophylactic (directed toward the convulsions) and specific (directed toward the mental defect). All the measures used for lessening the frequency of attacks should be made use of: the withdrawal of alcohol, a temperate diet (limitation of meat, the use of plenty of milk, vegetables, and fruit, the regulation of the bowels), attention to the heart and blood-vessels, and of the whole order of living. Probably an impending mental disturbance may be aborted by 10-14 grams (150 to 210 grains) of sodic bromid daily, taken in plenty of water, until bromin acne or bromism appear. Bromalin and bromipin may be used. To combat the states of excitation, isolation, baths and packs may be used, together with large doses of bromin, and hypnotics such as amyl hydrate, chloral hydrate, or 7.5 grains of isopral three times daily. At times even duboisin or hyoscin may be necessary hypodermically. Flechsig's Bromid-Opium cure is not suitable for the mental disturbances of epilepsy, and operative treatment is very questionable.

The Treatment of Epilepsy. For the treatment of epilepsy A. P. Ohlmacher[1] advocates the use of calcium salts. Alike to supplement the favorable record published by Dr. Littlejohn,[2] who shows by tabular exhibits the suppression in two patients of epileptic attacks by the administration of calcium lactate over a period of three or four months, and to secure such credit as may be due from the standpoint of originality, and historic priority, Ohlmacher reports his first case of epilepsy in which he used a calcium salt. The patient was 4 years and 4 months of age, suffering from attacks of both *petit mal* and *grand mal,* in excessive numbers. Thus in January, 1906, there were 844 attacks, 401 "light," 424 "hard," and 19 "convulsions." For the following months to the time of withdrawal of the bromid by gradual reduction, the attacks varied in number monthly from 530 to 1,012. "Following the withdrawal of the bromid, the usual change from numerous light to fewer severe attacks took place as shown by a monthly total of 35 to 118, and of these *grand mal* seizures numbered 34 to 73 a month. June 3, 1907, the treatment under special consideration was begun, and the record shows 18 attacks, all *grand mal,* for this month, none for July, 72 *grand mal* in August, 6 in September, and from Sept. 7, 1907, to the present writing, June 30, 1909, no further epileptic attacks.

"Mental Condition.—The normal mental growth at the outset of the epilepsy ceased abruptly, and retrogression

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(1) Jour. Amer. Med. Assoc., Aug. 14, 1909.
(2) The Lancet, May 15, 1909.

began, which, when I first saw the patient, had reached the stage of a low-grade epileptic imbecility. After the bromid was stopped the hoped-for mental improvement did not follow, and it was not until after all attacks had ceased with the inauguration of the new treatment that a gradual clearing of the mind was observed, and this unclouding is still going forward to a degree most striking after the apparently hopeless state that once existed. In fact, the child seems to have suffered a mental nonexistence for the two years and eight months of the epilepsy, and, with the suppression of the neurosis, to have resumed his mental growth from the time when the attacks began.

"*Treatment.* During March, April, and May the lad had frequent nose-bleed following the *grand mal* cycles, and the nurse claimed she could detect the odor of blood on the breath during a convulsion and prior to the onset of the visible hemorrhage. I was at this time actively engaged in the practice of therapeutic immunization where the problem of blood coagulability presented itself, and I had employed Wright's method of measuring the time of blood coagulation, as well as his plan of using calcium salts to fortify a defective coagulability. Accordingly, when my attention was arrested by the reports about the epistaxis, I made the first blood-clotting test, June 2, 1907, finding that the patient's blood failed to clot in 4 minutes, while a control blood under the same conditions firmly coagulated in 1½ minutes. The following day I began the administration of calcium lactate in doses of 7 to 10 grains dissolved in hot water and added to milk, three times a day. This medication has been continued with no change except occasionally reducing to one or two doses daily, to the present time."

The Surgical Treatment of Epilepsy in so-called idiopathic or hereditary cases is generally regarded as useless, and has been practically abandoned; but the experience of W. P. Carr[1] leads him to believe that such a conclusion is hasty. In the past ten years he has operated upon twenty cases, nearly all of long duration, but he does not go into any details as to the method of operating. He says: "The results have been most encouraging. Nearly all were either

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(1) New York Med. Jour., April 17, 1909.

cured or temporarily relieved to such an extent that they felt repaid for undergoing the operation. Eight have remained well to the present time, or were well when last heard from; one after nine years, one nine and a half years, one seven and a half years, one four years, one three and a half years, one for one year, and two for three months.

Speaking of one case upon which he operated, he says: "Believing there was some slowly developing lesion of the right cerebral hemisphere, probably a cyst or cold abscess, and regarding the case as absolutely hopeless without surgical intervention, I operated upon him at the Emergency Hospital, Sept. 21, 1908. A large osteoplastic flap was turned down from the right temporoparietal region, and the whole Rolandic area exposed.

"To my surprise, the brain was not edematous, but the tension was very great, and no pulsation could be felt or seen. A grooved director was carefully introduced in half a dozen different places and directions, in search of a tumor or cyst or abscess, but nothing was found. I was so sure there must be some growth or cyst to cause the great amount of tension, that I carefully introduced my finger into the fissure of Sylvius at least three inches, *and again into the substance of the occipital lobe, palpating the occipital lobe with one finger in the center of it and one on the surface.* But nothing was found. I replaced the flap, still feeling that there must be a growth in the brain, but not feeling justified in any further exploration."

[The italics are the Editor's. Surely such a procedure ought to be calculated to cause all sorts of violent occipital lobe disorders, but we are informed that the patient entirely recovered alike from the epilepsy, the operation, and the penetration of the occipital lobe to the core by the operator's finger!—ED.]

MIGRAINE.

Prodromes of Migraine. An address upon this topic was recently delivered by Sir William R. Gowers before the Westminster Division of the British Medical Association.(1) The premonitory symptoms, although sensory, do not include pain. They are restricted in range. The sense of sight is often involved, sometimes by loss, partial or general; sometimes by an elaborate display of its higher crude functions in light, color, and apparent motion. Other special senses do not participate, except the sense of touch. Neither smell, taste, nor hearing seems to be involved, although there is rarely a slight degree of vertigo, the precise nature of which is not clear. The involvement of cutaneous sensibility seems to be of touch, and is a frequent and marked manifestation. The arm is its most common seat. It may be felt in the tips of the fingers, passing along them, and going no farther, or as a zone of tingling around the wrist, which slowly ascends the arm, in the course of ten to fifteen minutes. As it passes up, it leaves behind it lessened sensibility, which gradually becomes normal, and with this some degree of loss of power, apparently the result of inhibition. After reaching the shoulder, the tingling hardly ever descends the trunk to the leg, although it may in rare cases be felt in the foot, and sometimes ascends the leg. Often, after ascending the arm, or primarily, it is felt at the mouth, in the lips, sometimes in the gums and tongue, on one side, but in the lips it is often felt on both sides. Here it is an intense tingling, sometimes compared to pins and needles, apparently as slight pricking, but it is never described as pain.

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(1) British Med. Jour., June 12, 1909.

"Still more frequently the premonition consists in interference with sight. There may be impairment of one-half of the field of vision, seldom if ever so complete as it is from organic disease, not reaching actually the middle line. Often the loss on one side is associated with a luminous spectrum, of which a typical form is that figured by Dr. Airey in the *Philosophical Transactions.* This begins as a star, a little to one side of the fixing point, which enlarges, becoming first an angled sphere and then a serrated or zigzag outline which breaks below, and the elements of which it consists become progressively smaller toward the free extremity. They are often colored, adjacent ones differing. Sometimes they are luminous without color. Often they are double. Within the zigzag, as it expands, there is a misty luminosity which becomes fainter internally, and hinders sight if an object is looked at. Thus the dimness of vision within the bright or colored zigzag outline resembles the numbness left by the tingling as it passes up the arm. After its expansion, the spectrum often ends by a whirling explosive appearance. In other cases an angled light appears near one lateral edge of the field, or both, sometimes several interlacing zigzags. Or a stellate bright object may appear on one side and remain, without expanding. In other cases a sort of luminous lacework occupies the middle of the field, sometimes bounded by a brighter band.

"This sensory disturbance may be always the same in some patients, in others varying. The premonition is often unilateral in character, and the succeeding headache is felt on the opposite side of the head. Sometimes the premonition is felt on both sides or has no lateral significance, and then it is succeeded by a medial or bilateral headache. These constitute the common rule, but exceptions are frequent; at least, a patient may fail to recognize a one-sided seat of the headache which succeeds a unilateral prodroma. The headache may be successively bilateral, that on one side ceasing when pain develops on the other side. Moreover, the premonition when unilateral may pass beyond the middle line in its course, although the headache is confined to one side.

"One other frequent symptom of the warning remains to be mentioned. When the sensory disturbance is right-sided it is often accompanied by impairment of speech of the character of aphasia. Sometimes the loss is almost complete; in other cases there is only a difficulty in the use of words.

"I may add another word regarding *treatment.* The benefit from nitroglycerin and strychnin, although often most conspicuous, is not invariable. Cases in which vasomotor disturbance is well marked externally sometimes derive no benefit. Why this should be the case it is not possible to say. Bromid taken regularly may then have more effect, or a small, long-continued course of salicylate. For the relief of attacks which are developing, a dose of antipyrin has often a quick influence, or Hochst's fused combination of antipyrin and caffein (migrainin), or a similar preparation made by Martindale, of antipyrin fused with salicylate of caffein (migralgin). Nitroglycerin may conveniently be given in the form of the 1 per cent. solution, liq. trinitrini, but the mixture containing it must be kept acid. It may therefore be usefully united with a digestive tonic. Dyspepsia is common in some of those who suffer from migraine, and an error in diet may give rise to an attack some time after the previous one."

AMBULATORY AUTOMATISM.

Ambulatory Automatism forms the title of a general review by R. Benon and P. Froissart.[1] [The article is here mentioned because of the interest taken in the United States in this subject of late, and the exceedingly complete bibliography which accompanies the review. Should any one wish to look up the subject, the references here set down, occupying one and a half pages, are so complete as to enable one to go into it exhaustively.—ED.]

Ambulatory automatism is, in mental pathology, a variety of *fugue,* not very infrequent, and interesting both clinically and medicolegally. A true *fugue* demands that two conditions be realized: (1) the subject must leave his home and not return to it, his psychic condition making it impossible for him to do so; and (2) his family and friends, without news of him, must consider him as disappeared, and be uncertain of his fate. Unless both of these conditions are fulfilled, there is no *fugue.* The onset is sudden, the duration more or less transitory, and the destination more or less accidental, leading to a sort of wandering. Often, before his departure, the subject has given no sign of any change in his mental faculties. During his fugue he is in a peculiar psychic state, which is not unconsciousness. When he comes to himself, he usually shows amnesia, which may be total. He passes abruptly out of the condition, just as he passed abruptly into it. The duration is one or more days, one or more weeks, and may last several months. While in the *fugue,* real wandering takes place, the patient traveling on foot, on the train, or by various means of conveyance.

It was Charcot who in 1888 created the term ambulatory automatism, but undoubted cases analogous to Charcot's had been previously reported by many other observers, most of which are here noted. A fairly complete record of cases reported since Charcot's illuminating *leçon* is also added. Very important is Heilbronner's article published in 1903,[1] based on 57 cases. His conclusions deserve quotation:

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(1) Gaz. des Hôp., July 31, 1909.

"(1) Among the individuals suffering from *fugues,* even if there be eliminated those whose wanderings are to be explained directly by permanent psychic disturbances, hardly a fifth part presents absolutely certain epileptic disturbances.

"(2) The *fugues* of epileptics are in no way to be distinguished from the nonepileptic (hysterical) *fugues,* neither by the behavior of the memory nor by any other special condition.

"(3) The number of individuals showing with their *fugues* signs of hysteria is much larger than the number showing signs of epilepsy.

"(4) One is not justified in making the diagnosis of epilepsy upon the symptom of a *fugue* alone or an analogous condition; and these conditions, coming on in seizures though they do, are not to be regarded as epileptic equivalents.

"(5) Fugue-states ought to be oftenest looked upon as the morbid reaction of degenerate individuals, against what may be called "dysphoric" sentiments.

"(6) The tendency toward *fugues* may become habitual.

"(7) Fugue-states, especially when of long duration, offer difficulties impossible of solution, even when there are undoubted epileptic antecedents.

"(8) A medicolegal appreciation of fugue-states should be based upon the habitual state of the individual rather than upon the *fugues* themselves."

Diagnosis. In a given case, the first thing to be decided is as to whether it is a *fugue* or not. It is a fugue if it fulfills the two conditions mentioned at the beginning of this paper, if it is associated with a peculiar psychic state, is abrupt, transitory, and accidental rather than purposeful, wholly without motive. And vagabondage is not the same thing as the fugue-state, it goes without saying.

If a *fugue* is diagnosticated, the next question is whether it is true ambulatory automatism or a case of dual existence. This is easily settled, for in the one case there is flight and wandering without purpose; in the other, there is an entirely different set of symptoms, the patient simply passing apparently into another character, without necessarily wandering. And if it be finally decided to be a case of true ambulatory automatism, the final question is as to its nature, whether hysterical, epileptic, alcoholic, etc.

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(1) Jahrb. f. Psych. u. Neurol., 1903, p. 107.

CHOREA.

Nature of Chorea. George M. Swift[1] insists that chorea is a symptom, not a disease. The idea that it is a disease-entity was due wholly to the English, in whose island it is apparently more prevalent than in the United States. The causes of incoördinated muscular movement are many, and occur in both old and young. "In the old we consider these movements when occurring just prior to death, in the course of an arteriosclerosis, as due to faulty nutrition and failure of action of the motor-control cells. The same sort of movements appearing in the course of a severe illness, a toxemia, or at a time of great stress, we also explain as due to faulty nutrition or lost action of the motor-control cells; as due to poisoning of them, or due to their exhaustion by overaction. Why, then, when such movements occur in children under certain conditions, should we regard them as due to something quite different?" Indeed, according to Swift, chorea belongs in the same category as "convulsions," "dropsy," and "jaundice."

Swift divides the cases now called chorea into several classes, depending upon the etiology. Class I comprises the many cases which occur in spring and autumn, most commonly in girls, between the ages of 7 to 14 years. A great proportion of these occur among those bright, nervously disposed children who strain to keep up their standing in school. These children become anemic, fatigued, irritable and exhausted, so that any little emotional shock, or, indeed, none at all, may precipitate an attack manifested by uncontrolled movements. Class II comprises a large number of cases due to infections, such as malaria, pneumococcic and rheumatic infections. This explains why so many kinds of bacteria have been isolated in chorea. These

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(1) Amer. Jour. Med. Sciences, September, 1909.

cases are prone to endocardial and pericardial complications. Class III comprises the grave cases, now called chorea major and chorea insaniens, resulting fatally. These cases Swift believes to be really cases of septic or malignant endocarditis. Though he has had but two cases in his experience, both showed *postmortem* septic endocarditis. B. Sachs reported two such cases under the title "Grave Chorea and Its Relation to Septicemia."[1] Class IV comprises the cases now called Huntington's chorea. If one looks upon the disease as due to arteriosclerosis affecting the vessels of the motor cells, one removes everything unusual about it; for there is nothing especially strange about the fact that some families have a tendency to degeneration of a certain set of cerebral blood-vessels. Class V comprises the cases of chorea gravidarum, and no one questions the idea that this so-called disease is due either to a toxic state or to nervous depression in a pregnant woman.

The author concludes that choreic movements when occurring in the young are symptomatic only; that our teaching concerning so-called chorea should be broadened; that chorea is not a disease *per se* to be treated as such, but rather that the cause of the choreic symptoms should be carefully sought in every case; and that treatment of the condition should be directed to the cause alone, not to the resulting symptoms.

Respiration in Chorea. A study of the respiratory signs of chorea minor has been made by W. W. Graves.[2] "Having rendered the patient as easy and comfortable as possible, the following respiratory phenomena may be observed, and confirmed by tracings of the respiratory movements:

"1. Irregular breathing. This is probably due to two factors: (a) Lessened amplitude in some respiratory movements and increased amplitude in others, giving one the impression that no two respiratory excursions are equal. (b) Sudden halts—cogs occurring either during the inspiratory or expiratory phases, and most frequently during the latter, but rarely more than one cog occurring in any phase. These do not occur in every respiratory movement, but are of frequent occurrence and appear to be due to a

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(1) See Practical Medicine Series, 1908, Vol. X, p. 61.
(2) Jour. Amer. Med. Assoc., Jan. 30, 1909.

sudden arrest of the movement in one phase by the development of an incomplete movement of the opposite phase, but the original movement usually goes on to completion. These cogs render the respiratory movements distinctly jerky, and may well be called choreic inspiratory and expiratory cogs. An inspiratory cog occurs most frequently near the end of expiration and an expiratory cog near the end of inspiration.

"2. Now and then a sudden deep inspiration occurs, far exceeding in amplitude the usual unequal inspiratory movements, and is succeeded by a sudden explosive (forced) expiratory movement, the whole differing essentially from a sighing respiration by its rapidity in its every phase and by the absence of a pause at the end of expiration.

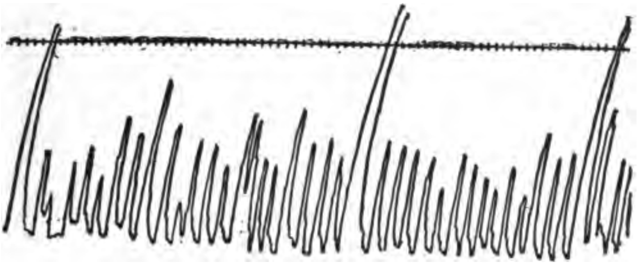

Fig. 1.—Respiratory tracing in Case 1 (J. M.), made June 6, 1907. Showing inequality of respiratory excursions, inspiratory and expiratory cogs, and frequent sighing respirations; respiratory rate 34 a minute.

"3. Relative decrease in the length of the normal expiratory phases and relative increase in the length of the so-called normal expiratory pauses: so that the time of an expiratory phase may become equal to or less than the time of an inspiratory phase, and distinct pauses are of frequent occurrence at the end of expiration.

"4. Sudden changes from abdominal to costal, or from costal to abdominal breathing, occur frequently, both being observed at times in a single respiratory excursion, giving one the impression, for the moment, of a combination—a mixture of abdominal and costal breathing, and the frequent impression of incoördination—a dissociation in the respiratory movements. When "pure abdominal breathing" occurs in chorea, movements of the whole chest are reduced to a minimum, and when "pure costal breathing" occurs the upper abdomen remains practically motionless.

"5. The respiratory rate is frequently accelerated, and occasionally this may be slower than normal."

The accompanying figures show respiratory tracings from two cases of chorea.

An elaborate review of the literature upon respiratory changes in chorea minor accompanies the article. A consideration of the literature "warrants the conclusion that certain respiratory phenomena are among the constant signs of the disease. This conclusion finds confirmation in the motor manifestations of the disease other than the respiratory. The involvement of the trunk and abdominal muscles are among the earlier manifestations of the disease and among the last to disappear. In early and active stages, the careless, sluggish, variable attitudes, and shifting, writhing movements of the trunk, are due to the spontaneous choreic contractions in these muscles. Careful inquiry concerning the first change noted will elicit statements of sluggish, variable attitudes—'inability to keep still,' etc. Inspection of the naked trunk during the early and active stages of the disease will show that the variable attitudes are incident to or immediately follow visible and

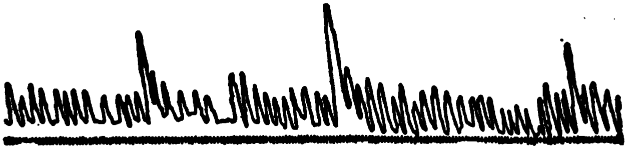

Fig. 2.—Respiratory tracing in Case 2, made June 12, 1907, showing inequality in respiratory excursions, sighing respirations, respiratory cogs, expiratory pauses and lessened ratio between inspiration and expiration; respirations about 19 a minute.

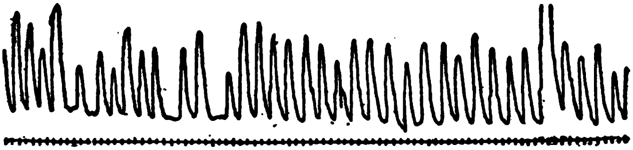

Fig. 3.—Respiratory tracing in Case 2, made June 19, 1907, after six days rest in bed. Respirations 28 a minute.

palpable contractions in the muscles just mentioned. After the disappearance of all the general and the usually recognized motor manifestations of the disease, contractions in an attenuated degree persist in these muscles, and are among the last of the motor phenomena to disappear."

Diagnosis. The differential diagnosis between chorea and tic is considered by H. T. Patrick.[1]

"The movements of chorea are involuntary and not amenable to control by the patient. Those of tic, however bad, are always volitional, even if not voluntary, and may always, at least for a short time, be controlled by the will. As a rule the patient with tic says he 'can't help it,' and it is perfectly true that the tic movement often is unconscious, just as the man with the fixed habit of profanity swears involuntarily and unconsciously; but in the one case, as in the other, it is always possible to awaken consciousness and inhibition. In the presence of a clergyman or of ladies, the most profane man will talk for an indefinite time and never utter an oath. Many a time I have sat by a youngster with tic, holding a pin in my hand, and, the child understanding that the result of a jerk or twitch on his part would be a jab with the pin, I have waited long in vain for the movement which was accustomed to come every minute or less. This maneuver never succeeds in chorea.

"In chorea the movements are incoördinate, purposeless; pathologic in the association and sequence of muscular contraction. They are grotesque because of their incoherence and disorder. In tic the movements are coördinate and purposive; physiologic in the association and sequence of muscular contraction. They are grotesque only because malapropos.

"On the other hand, the movements of tic are coördinate and such as well persons often execute. They are movements adapted to an end, a natural and ordinary end, only an end which is no longer there. They are unnatural in frequency of repetition without obvious cause, but from looking at a single movement one could not know that it is pathologic; its aspect is normal. For instance, a frequent tic in children is a peculiar shrug of the shoulder

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(1) Jour. Amer. Med. Assoc., May 1, 1909.

involving the arm, such as a nervous or sensitive child makes when the armhole is too tight. When there is no irritant from the clothing, no physical cause for the shrug, when the movement is oft repeated, uncontrollable, and has become a tic, it still preserves its coördinate and purposive character. It looks the same; but it may become greatly exaggerated, pathologic in its excess.

"In chorea, as the movements are entirely disordered and neither have nor have had any object, no two movements in succession are just alike. In tic the same movement is repeated over and over.

"When choreic movements grow worse and spread, they do so without relation to anatomic or physiologic groupings, except that in the beginning and in mild cases they are apt to be limited to one side of the body. A tic extends in one of two ways. Ordinarily it spreads by involving adjacent and functionally related groups; by adding correlated physiologic acts. A tic of the nose is apt soon to involve the lips and adjoining facial muscles. The sounds of snuffing or sniffling are produced, and if the disease grows worse pharyngeal or laryngeal sounds may be expected, with strong inspiratory or expiratory efforts."

Chorea Gravis. A case of chorea gravis with postmortem examination is reported by H. Mèry and L. Babonneix.[1] The patient, a little 4-year-old girl, had had measles six weeks before. On entering the hospital the chorea was not severe, but it grew rapidly worse, and within four days an erythema resembling that of scarlet fever appeared. In the last two days of her life of seven days in the hospital the choreic movements became excessive, and the temperature rose as high as 104° F. The autopsy showed tuberculosis in the bronchial glands, lungs, spleen, and peritoneum, a mitral insufficiency with fresh vegetations, and changes in the brain. Macroscopically the latter was intact, but microscopic examination of the cortex showed (1) a slight rarefaction of the superficial layers, (2) some dilatation of the perivascular sheaths, and (3) some small hemorrhagic foci. There were no "chorea bodies." The greater part of the cells in the optic-striate region were involved. Some had completely lost their chromatophile bodies, others had lost their prolongations; others had eccentric nuclei. In the Rolandic area the small pyramidal cells were involved in a perinuclear chromatolysis; some had lost their processes; others were surrounded by neuroglia. Most of the large pyramidal cells were also involved. In general the lesions were diffuse.

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(1) Gaz. des Hôp., Sept. 15, 1908.

Many cases of tuberculosis as an antecedent of chorea have been reported and are reviewed in the present article. The appearance of a scarlatiniform erythema in chorea is not exceptional, other cases having been reported. The literature on the pathology of chorea gravis is also reviewed. Worthy of emphasis is the absence of "chorea bodies," and the fact that there were found undeniable lesions of the greater part of the large pyramidal cells, of the cells of the central gray nuclei, and of the pons. These lesions were *not* artefacts, the technic of preparation being scrupulously exact.

Treatment. The treatment of chorea is described by G. Köster,[1] after a few preliminary statements as to etiology, in which he affirms that infection and fright are both potent factors. (a) Salicylic acid preparations, sodic salicylate, aspirin, salol, etc., are of no avail, even though chorea is infectious. (b) The bromids have been recommended, especially sodic bromid, in doses of from 15 to 45 grains daily, according to the age of the child, or of bromglidin, in doses of from 7.5 to 22.5 grains per day. To the bromid, an iron preparation may be added to overcome the anemia present. (c) Stronger sedatives, such as opiates, morphin, and chloral hydrate, are discountenanced by Köster, but small doses of bromural or veronal are often of value without being detrimental. (d) Arsenic in the form of Fowler's solution is the best remedy. For a 7-year-old child he prescribes 7 parts of Fowler's solution to 13 parts of water, and gives of the mixture 5 drops three times daily, increasing a drop every three days to a maximum, and then correspondingly diminishing the dose until it is back to 5 drops. If arsenic at first disagrees, he stops it, and when signs of irritation have disappeared,

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(1) Deutsche med. Woch., Jan. 7, 1909.

he begins it again. Often it is tolerated on the second trial. Of late, atoxyl has been recommended, but Köster regards it as too toxic to be trusted.

Of external therapeutic measures he regards (a) electricity as entirely useless, whether the galvanic or faradic current is used. (b) Gymnastic exercises, on the other hand, he regards as very valuable, but they must be perfectly adapted to the case. Classes for gymnastic exercises exclusively for chorea are therefore useless, and even harmful. (c) Massage with gymnastic exercises, is an excellent combination, and should be used in all cases. (d) Hydrotherapy is indispensable, in the form of hot rubbings of the whole body (sponging with water at a temperature of 105° to 115° F., followed by rest in bed), and warm tub-baths at 96° F. from half an hour to an hour in duration. Sea-baths are often useful. No treatment is of value which does not improve the general condition of the patient, and build up the body. The milk-cure may be advantageous; at any rate the avoidance of coffee and alcohol is important. The diet should be a mixed diet, and a nutritious one.

The treatment of Sydenham's chorea by the injection of solutions of sulphate of magnesia into the spinal canal is advocated by G. Marinesco.[1] The old methods, such as the use of arsenic in any form, especially sodic cacodylate and disodic methylarseniate, antipyrin, hyosin, and exalgin, leave much to be desired. Marinesco was therefore led to try sulphate of magnesia in 25 per cent. aqueous solution, special care being taken to obtain absolutely pure magnesic sulphate. Marinesco's idea came from an article by S. J. Melzer[2] who found that salts of magnesium injected into the subcutaneous or intra-arachnoidean spaces, exert a marked anesthesia in the territory supplied by the nerves involved. Thus 1 c. c. of a 25% solution of magnesic sulphate within three or four hours causes paralysis and anesthesia of the lower extremities and pelvis. It is about a week before motility and sensibility are normal. In some of Melzer's and Auer's cases retention of urine ensued for one or two days. Calcaterra, noting

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(1) La Semaine Méd., Nov. 18, 1908.
(2) Med. Rec., Dec. 16, 1905.

the inhibitory action, has used magnesic sulphate intravenously.

Marinesco reports four cases which he has treated by sulphate of magnesia injected into the spinal canal. The sulphate of magnesia must be pure and not effloresced, 25 grams being dissolved in 100 grams of water. In general as much cerebrospinal fluid is withdrawn as one wishes to inject of the solution of sulphate of magnesia. Without going into unnecessary details, it may be stated that the cases of chorea were typical ones of more than average severity, the cases being detailed at length. The doses given varied. Thus the first patient, a girl 14 years of age, received 3.5 cubic centimeters at her first injection. At the end of forty-five minutes the choreic movements had diminished a good deal, but still remained the next day. Three weeks or so after the first injection, the same amount was again introduced. Within three days' time the choreic movements entirely disappeared. The second patient, 22 years of age, was given 5 cubic centimeters. An hour later the patient felt numb in her legs, and she could not move them. Three hours afterward she was somnolent, the respiration 34 per minute, the pulse 106. The numbness and paresis gradually disappeared, and all traces of chorea had meanwhile been swept away. These two cases may serve as types of the other two, and as examples of dosage. Marinesco affirms that the new treatment shortens the duration of the disease considerably. The sedative action of sulphate of magnesia so given is truly remarkable, not more than two injections ever being required. The treatment is, however, not without its inconveniences, which may sometimes be serious, respiratory accidents being occasionally encountered. In experimental work previously Marinesco had met with sensory and motor disturbances and sometimes urinary disturbances. None of these accidents took place, however, in the four cases here reported. The author is inclined to attribute some of the accidents to drug impurities. Only Sydenham's chorea is benefited by this method of treatment.

Chorea is carefully described by Guthrie Rankin,(1) nothing new being mentioned in the symptomatology, however. He gives as the proximate cause a microorganism similar to that of rheumatism, the toxins of which are responsible for the nervous manifestations. It must be admitted, however, that the direct excitant of an attack is in many instances some form of emotional disturbance.

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(1) British Med. Jour., Sept. 12, 1908.

"In the treatment of the disease, rest, both mental and physical, is of special importance, and is indicated in every case, no matter how mild. In many instances the child will recover under the influence of rest alone without any medicine whatever, but many cases will get steadily worse if not treated by drugs. The child ought to be put to bed and kept there until all movements have ceased, so as to minimize the risk of cardiac complications and at the same time expedite the natural tendency to recovery. The bowels must be kept gently open by occasional doses of mild alteratives. There is no specific for the disease, but better results are got from arsenic than from any other drug. It ought to be administered from the earliest development of symptoms, in moderate doses at first, but judiciously increased and pushed up to the limit of toleration. For a child 8 years old, Fowler's solution may be commenced in 3-minim doses, and gradually increased until within a week 10 minims are given three times a day. Children bear arsenic well, and can frequently stand large doses without inconvenience. Brilliant results have been claimed for heroic doses of from 15 to 25 minims, given from the onset of symptoms, three times a day after meals, in a large tumblerful of alkaline water. It is doubtful whether the ordinary and safer dosage of from 3 minims gradually increased to 10 is not attended, in most instances, with perfectly satisfactory results, without the disadvantages of the need for constant medical supervision. When the stomach is intolerant of the drug, Eulenberg has advocated its administration hypodermically, but in children the emotional upset which results from the prick of the needle constitutes a serious drawback. Cacodylate of soda is extensively used in France instead of liquor arsenicalis in the treatment of chorea. It may be given either hypodermically or in the form of a pill in much larger doses than other preparations of arsenic, but the garlic odor it imparts to the patient's breath is a strong reason against its administration. Chapnis has shown experimentally that arsenic is less toxic when combined with butter than when given in solution. A known quantity of arsenious acid, according to the dose to be administered, is taken and added to chlorid of sodium in the proportion of 0.1 gram of the sodium salt to 0.005 gram of arsenious acid. This mixture is triturated with 10 grams of fresh butter, and given, spread on bread, after a meal.

"Besides arsenic, other drugs may be called for, according to the indications in each case. Thus, anemia is frequently an important accompaniment and demands the administration of iron for its relief; nutrition may be much impaired, and will be benefited by the addition to the daily dietary of maltine and cod liver oil; if rheumatic symptoms are pronounced, the salicylates must be given in suitable doses; excessive restlessness demands the exhibition of bromids; and so on. If restlessness is accompanied by a dry state of the skin, diaphoretics are often very soothing. It is under such circumstances that antimony is specially useful. When exhaustion threatens, stimulants are necessary, and should be given in quantities to suit the requirements and age of the patient.

"In all severe cases something should be done to control the muscular movements until the more slowly acting drugs above referred to have time to exercise their power. The movements may be so incessant as to threaten the patient's life from exhaustion, and under these circumstances more good will be got from hydrate of chloral than from any other drug. Gairdner declared that, when pushed sufficiently far, it has an almost absolute power of suspending or controlling the muscular disorder. It may be given either alone or combined with bromid of potassium. If, for any reason, chloral is inadmissible, sulphonal or trional may be tried.

"During convalescence, tonics, a generous dietary, carefully graduated gymnastic exercises, and change of air are all serviceable. The child should not be allowed to undertake mental work until complete recovery is established."

DISEASES OF THE BRAIN AND MENINGES.

EPIDEMIC CEREBROSPINAL MENINGITIS.

Pathology. Two cases of multiple encephalitis in cerebrospinal meningitis of the epidemic sort are reported by Maschke.[1] In each of them at the postmortem examinations numerous foci of encephalitis were found, in one case these foci predominating even over the lesions of the meninges themselves. Such cases are rare, being said to be very rare by Albrecht and Ghon, and infrequent by Weichselbaum and Kutscher. In the first case here reported a purulent exudate was found spread over the base of the brain, and to some extent over the convexities. The brain substance was of moderate consistency, and contained multiple grayish red, small, fresh encephalitic foci, which had developed perivascularly. The diplococcus intracellularis was found in sections through these foci. The findings in the second case were those of an incipient cerebrospinal meningitis, with multiple foci of encephalitis well developed, varying in size from a hemp-seed to a bean.

Prognosis. Concerning the prognostic value of agglutinins and opsonins Alexander Macgregor[2] may be quoted as follows: "It was found that agglutinating power had a definite relationship to the clinical features of the disease. This point is accentuated by considering the cases in the following groups:

"*Agglutinins.*—A. Of 21 acute fatal cases which died within the first fortnight, in 14 agglutinating power was absent. In 7 cases which died on days varying from the fifth to the tenth, some agglutinating power was present, varying from 1 in 10 to 1 in 80. Clinically, this type has an abrupt onset, with headache and vomiting, and unconsciousness rapidly supervenes. The symptoms are those of a very intense general infection. *Postmortem,* there is found a general or advancing leptomeningitis.

"B. Forms a class in which high agglutinating power was associated with an early and decided clinical reaction.

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(1) Berl. klin. Woch., Aug. 24, 1908.
(2) British Med. Jour., Oct. 31, 1908.

The onset is acute, as in Group A. Although the symptoms are scarcely less grave they differ from those in the preceding group in the fact that they terminate somewhat abruptly by crisis. This favorable termination might occur on any day up to the tenth, but usually on the seventh or eighth. In certain of these cases agglutinating power reached its highest, 1 in 80 or 1 in 100. Such cases may either convalesce or progress to a chronic stage.

"C. Twenty-one cases in which agglutinins were never found (serum dilution 1 in 5, negative). These cases were all characterized by the absence or very short duration of acute initial toxic symptoms; 11 of these recovered.

"D. Twenty-one cases whose serums possessed a variable agglutinating power. These represented a type intermediate between Groups B and C.

"The following conclusions may be based on the results:

"(1) In very acute infections, where toxemic symptoms are severe and death rapidly ensues, there is an entire absence of agglutinins from the blood. These cases form the chief epidemic type. (Group A.)

"(2) On the other hand, in cases which are chronic from the outset, the blood contains no agglutinins. These cases may have prolonged pyrexial courses. (Group C.)

"(3) The degree of agglutinating power which is developed in any given case depends on the initial features of the case, being proportional to (*a*) acuteness of onset, (*b*) duration of primary toxemia, and (*c*) degree of reaction. (Group B.)

"(4) Agglutinins are produced only in response to the early toxic phenomena; they appear to be independent of the subsequent course of the case.

"(5) A number of acute cases, lasting from two to three days, came under notice—4 out of 6 cases of this type recovered. In these cases agglutinins were absent or very feeble, hence they are of little value in the diagnosis of such short, often indefinite, cases. It is obvious that some method of diagnosing such cases after recovery would be of great value from an epidemiological standpoint.

"*Opsonins.*—In this study the method followed was that of Wright and Douglas, and the precautions mentioned by Houston and Rankin were in the main confirmed.

"The behavior of the opsonic index in 55 cases (175 observations) embracing the different types and periods of the disease may be described as follows:

"1. The highest indices occur mostly in those cases with acute initial symptoms of not too short duration which also, as has been noted, possess most marked agglutinating power. Where agglutinating power has been low the index has usually been low.

"2. Accordingly a high index is a sign of forcible reaction to a fairly severe infection and is of no prognostic value, as cases with low index may recover and cases with a high index have died.

"3. The indices tend to be highest during the second and third weeks of illness in an average case.

"4. The degree of immunity after recovery, as measured by the opsonic index, is very variable. There may be an active opsonin in the blood for many weeks after resolution or it may occasionally disappear comparatively rapidly.

"5. It is scarcely worth while to refer to its diagnostic value. In a typical, severe case the opsonic films are no doubt extremely characteristic, but the isolation of the meningococcus from the cerebrospinal fluid has in the writer's experience proved the most valuable and uniform diagnostic method.

"In conclusion, it may be said that observations on agglutinins and opsonins are thus far of uncertain value in regard either to prognosis or therapy. Prognosis in the individual case depends upon factors not yet ascertained. The infective process itself may be so acute as to cause a fatal termination, or the exudate may be resolving satisfactorily, and yet the patient finally succumb to hydrocephalus, which has been brought about, possibly, by organization changes that accompany the healing stage—that is to say, treatment may be undertaken at a time when profound pathological changes are in progress which it may be impossible to modify."

The *sequels* of cerebrospinal fever of the epidemic variety have been studied by L. Cohn,[1] his material consisting of 82 cases seen during the years of 1905, 1906, and 1907, in Posen, every case being proved bv bacterial

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(1) Berliner klin. Woch., Jan. 11, 1909.

examination. Of these 82 cases 50 died, leaving 32 who left the hospital for the most part entirely well or satisfactorily convalescing; but of these a child died soon after leaving, of a secondary hydrocephalus. Three were lost sight of, and another died of diphtheria subsequently. Thus 27 cases were investigated carefully.

These cases fall into three groups: (I) Cases, 8 in number, of a mild course. These all healed without showing a single departure from the normal, the special sense organs being entirely intact. (II) Cases, 15 in number, of moderate severity. Of these, a part were found perfectly free from sequelæ. In others paralyses lasted for various lengths of time, as an abducens paralysis lasting six months. Of four adults in the group, one complained of persistent headache, and three of paresis in the arms and legs. Three also complained of a weak memory. One case, a man 31 years of age, developed hydrocephalus after four weeks of perfect convalescence. (III) Cases, 4 in number, of great severity. Three of these showed complete deafness, in 2 of which there was facial paralysis, but this disappeared, leaving deafness alone persistent.

Interesting is the study of the agglutination test in 3 of the 4 latter patients, one of them showing the test 2¼ years after the onset of the disease, a second 2⅛ years, and the third 1¼ years afterward. The second case showed agglutination in 1 to 1,000 dilution. The author's conclusions follow:

"1. In all mild cases a complete restoration to health, in the clinical sense, follows.

"2. Even in severe or extremely severe cases a complete restoration to the normal is possible. The likelihood of a cure persisting is greater in children than in adults. In adults disturbances were still noticeable 2½ years after the illness.

"3. Even after four weeks of apparent convalescence hydrocephalus may appear.

"4. The complication most to be dreaded is deafness. While all paralytic phenomena tend to regression, even inactivity of the pupil, deafness, once established, persists.

"5. The agglutination test by the blood serum for the meningococci was in two cases positive more than two years after the disease."

Serotherapy. (a) *Flexner and Jobling Serum.* The serum treatment of epidemic cerebrospinal meningitis is discussed by L. W. Ladd,[1] based upon his experience in 31 cases, 11 of whom died, and 20 of whom recovered, a mortality of 35.5 per cent. He first used the serum April 2, 1907, in a community of 600 inhabitants, among whom 18 cases appeared in three months. Of these 12 died and 6 recovered. In 3 cases only, serum was used, and all recovered. In the other 15 it was not used, and all died. In the earlier cases 5 to 10 c. c. of the serum were used, but in the later cases 15 to 30 c. c. at a time, provided that an equal amount of cerebrospinal fluid was withdrawn. In some instances two injections a day were made. When the meningeal symptoms disappeared and the temperature subsided, the further use of the serum was discontinued until a rise in temperature or increase in meningeal symptoms made it seem advisable again to employ it. In 30 of the 31 cases the characteristic Gram-negative *Diplococcus* was found. In one case, clinically a typical one, only a few drops of bloody serum were obtained by lumbar puncture, from which nothing definite could be made out. The cases seen were not mild ones, but rather cases of marked severity, taxing to the utmost the therapeutic value of the serum.

Twenty-four cases were treated 96 or less hours after onset, and of these 16 recovered. Four moribund patients appear in the group. Eighteen cases were seen 72 hours or less after the onset, and of these 14 recovered. Thirteen cases were seen 48 hours or less after the onset, and of these 9 recovered. Five cases were seen 28 hours or less after the onset, and all recovered. The average duration of the disease in cases of recovery when seen 28 hours or less after the onset of symptoms, when treated with the serum, was 12 days; above 28 hours and including 48 hours, 19 days; above 49 hours and including 72 hours, 49 days.

"In most cases in which recovery takes place after the injection of serum the spinal fluid progressively becomes clearer and diminished in amount. In several cases, especially those in which few or a moderate number of menin-

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(1) Jour. Amer. Med. Assoc., Oct. 17, 1908.

gococci were found, these could not be cultivated subsequent to the injection of serum, though they grew readily at first. In such cases, as a rule, the diplococci do not stain well. At other times growths could be obtained until after the second or third dose of serum had been administered, and occasionally when considerable amounts had been given after longer intervals of time had elapsed."

Frank J. Sladen[1] discusses the same subject, recounting his experience with Flexner's serum in Baltimore, in the Johns Hopkins Hospital. Of the 33 patients treated by the old method of lumbar puncture in previous years, 21 died and 12 recovered, a mortality of 64 per cent. Of 21 cases treated during the 5 months preceding this report, by means of Flexner's serum, the mortality was only 14 per cent., only 3 of the 21 dying. One of the three was a fulminant case. The second had a complicating bronchopneumonia of both lungs, dying 48 hours after admission; and the third was a boy 29 months, untreated until the fourteenth day, being received into the hospital practically *in extremis*.

"It has been our rule to do a lumbar puncture as early as possible in every case with symptoms suggesting meningitis, and to inject serum, giving the patient the benefit of the doubt. By this routine one patient with pneumonia, one with typhoid fever, two with pneumococcal meningitis, one with influenzal meningitis, and six with tuberculous meningitis have received serum. In no one of these cases has any bad effect resulted.

"It has taken from twelve hours to three days to reap the benefit from the serum. All favorable cases react to a certain extent after one injection. Some cases need but the one injection. Two cases have required six injections and one seven. We believe the serum should be given daily until the drop in temperature and the disappearance of symptoms and signs indicate that the process is arrested. And it should be renewed at any sign of flare-up.

"1. The effect on the *temperature* has been to cause a fall in one of two ways—either abruptly or by lysis. Eight cases terminated abruptly and eight by lysis. In the other five the temperature remained elevated—in two due to

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(1) Jour. Am. Med. Assoc., Oct. 17, 1908.

complicating pneumonia. The other three were fatal. One case remained normal in temperature after a single injection, but the majority showed a rise the next day and required from two to six injections to preserve a normal temperature.

"2. With the fall of temperature, in three to twelve hours after the first injection, the *symptoms and signs* begin to clear. The symptoms—delirium, headache, pain in the neck and back, and the general hyperesthesia—disappear with remarkable rapidity. The signs—strabismus, stiffness of the neck, retracted head, opisthotonos, and the Kernig sign—are more persistent. The Kernig is often the last sign to disappear. The result is the unusual picture of a child, bright, cheerful and alive to its surroundings, but with a stiff neck and back, occasionally in opisthotonos.

"3. The effect of the serum on the *spinal fluid* itself is exceedingly interesting. Three changes are brought about:

"First, the polymorphonuclear leucocytes are increased in the spinal fluid. This is only after the first injection, and they disappear in the process of healing. From 200 cells to the cubic millimeter they have been increased to as many as 4,000. The influence of this on the leucocytes in the circulating blood is striking. A high leucocytosis, 30,000 to 40,000, is reduced by the demand in the spinal fluid; a low one, 16,000 to 18,000, is increased.

"Second, the meningococci, both extracellular and intracellular, before serum, become entirely intracellular, so that it is often difficult to find organisms outside of cells in specimens after serum.

"Third, as they become intracellular they are being destroyed. They no longer stain deeply and distinctly, but are ill-defined and hard to see. They resemble granules rather than clear-cut diplococci. This is evidenced by the cultures. The number of colonies from approximately the same amount of centrifugalized sediment are reduced from 200 to 300 down to 8 or 10 after one injection of serum. Most cultures have been sterile after two injections, several after one.

"In conclusion, from this small experience, we may briefly summarize our impressions as follows:

"1. All cases of meningitis in which meningococcus infection is suspected should undergo lumbar puncture and serum injection as early as possible.

"2. The serum does no harm in cases not of the meningococcal type. It may do good.

"3. The course of the disease is changed by the serum. The long-drawn-out chronic cases are not seen, and the terrible sequelæ—often worse than death—are rare.

"4. As to the character of the serum, the rapidity with which signs and symptoms disappear suggests an antitoxic property; the positive chemotaxis for polymorphonuclear leucocytes, and the promoted phagocytosis are most definite and constant features; finally, the reduction in the number of diplococci, the change in their staining properties, and the loss of viability speak for a bactericidal power—although it is a question if this may not be explained by the phagocytosis."

The serum treatment and the prognosis, under various forms of therapy, of cerebrospinal fever, is also discussed by H. Koplik.[1] He says: "I have thus far treated thirteen patients with the serum, two of which are under observation, two of which died; the remainder recovered with the exception of one, which was discharged improved. A résumé of these cases will be given by Dr. Flexner in his article on the serum, and they are to be found under the heading of cases coming from the Mount Sinai Hospital.

"I think it would be rather premature and unfair in every way to draw any conclusions as to the serum until we have seen a larger material and until we have tried it in an epidemic of the violent type, such as we passed through in 1904 and 1905. On the other hand, it may be said that the serum of Flexner not only makes a very favorable impression, but is certainly a factor which cannot be excluded from the therapy of cerebrospinal meningitis, no matter what our subsequent conclusions may be. It certainly does appear that with the serum as we perfect it we may have an improvement in the percentage of recoveries.

The treatment of cerebrospinal meningitis by the serum of Flexner and Jobling, based upon 442 cases, is discussed by L. Emmett Holt,[2] ten typical cases showing the effects of the serum being reviewed, and temperature

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(1) Med. Record, Oct. 3, 1908.
(2) British Med. Jour., Oct. 31, 1908.

charts presented. "The serum is obtained from horses which have been immunized by injections derived from many strains of the meningococcus. Between four and five months are required before the serum of a horse is strong enough for use. The serum has been distributed from the Rockefeller Institute, accompanied by some general suggestions as to its use.

"*How the Serum Acts.*—Dr. Flexner states that the serum acts chiefly upon the meningococci, diminishing their viability and increasing their capacity for phagocytosis. By its effect upon the microörganisms it arrests the inflammatory process. To accomplish this result the serum must be brought directly into contact with these organisms and in a certain degree of concentration. This helps one to understand why the serum is practically without effect when given subcutaneously, also to appreciate the advantage of withdrawing by puncture as much fluid as possible before the injection is made, and the necessity for full doses early before important lesions have occurred.

"While, then, essentially a bacteriolytic serum, it must possess some antitoxic value as well; for in no other way can be explained the striking rapidity with which the subjective symptoms often improve after injection.

"*How the Serum Is Used.*—In a case presenting the symptoms of cerebrospinal meningitis a lumbar puncture should be made at the earliest possible moment. If the fluid withdrawn is purulent or cloudy, to save time, the serum should be injected at once, without waiting for the result of an examination of the fluid. It is quite true that in a small proportion of cases the disease will be found to be due to the pneumococcus, or, still less frequently, to some other organism than the meningococcus. This has happened in a few instances, but no harm was done nor does any seem likely, though, of course, no benefit is to be expected.

"In making the puncture the Quincke needle is to be preferred to the exploring needle often employed. It is desirable to withdraw all the fluid that will come away readily after raising the body to the semi-erect position and flexing the head upon the chest. The serum, previously warmed to the body temperature, is injected through the puncture needle without withdrawing the latter from the spinal canal. This should be done very slowly, the procedure usually occupying several minutes. The entrance of the serum is facilitated by elevating the hips and lowering the head. It need hardly be said that the entire operation of puncture and injection should be carried out with the strictest aseptic precautions. While it is desirable to withdraw considerable fluid, it is not absolutely necessary that as much be removed as the amount of serum to be injected. Late in the disease it is often necessary to inject much more, always with caution, however, and one may even inject after a 'dry' puncture, being, of course, sure that the needle is in the spinal canal.

"The question of dosage is not yet definitely settled, and much larger experience will be required before it can be. The initial dose at present advised for a severe case is 30 c. c., to be repeated in twelve hours if no improvement is seen, and thereafter daily until the temperature falls and the nervous symptoms abate. For the milder cases a dose of 15 to 20 c. c. has often been found to be sufficient, and in many cases a single injection only has been necessary. The object of the injection being to arrest the disease by acting upon the bacteria which are causing it, it follows that considerable quantities must be introduced at one time if it is to be effective. A few full doses near together may control the disease; while the same amount of serum administered in smaller quantities and distributed over a longer time may accomplish very little.

"My own feeling is that future experience will establish the desirability of using considerably larger doses than have hitherto been employed, especially at the outset of the disease, and where the amount of fluid removed is large.

"*The Effects of the Serum.*—Marked changes take place in the cerebrospinal fluid after the injection of the serum. In twenty-four hours the meningococci are much reduced in number and their vitality lessened. They are so altered that they stain with difficulty. After two injections, if not separated by more than a day, it is generally impossible to grow them, although they are usually present in small numbers. The number of polymorphonuclear cells in the fluid is much reduced, and there is seen coincidently a marked fall in the number present in the blood. All of these phenomena point to a decided reduction in the inflammatory process. Within twenty-four hours an effect is usually seen upon the symptoms; this is naturally most striking in the cases injected early. The temperature often falls 4 or 5 degrees, and may not rise again. The nervous symptoms—delirium, stupor and hyperesthesia—may rapidly improve and consciousness return. At the same time the patient's general condition, as shown by his pulse, respiration and prostration, is distinctly better. The last symptoms to disappear are usually the stiffness of the neck and the rigidity of the extremities. Occasionally skin eruptions, most frequently of an urticarial character, are seen, as after the use of diphtheria antitoxin.

"*Results of the Serum Treatment.*—The value of the serum treatment may be determined by a consideration of three points: (1) The mortality of the cases treated; (2) the duration of the disease in the cases which recover; (3) the frequency of complications and sequelæ, or the completeness of recovery.

"Dr. Flexner has kindly permitted me to report for him to this Association the results of 442 cases returned up to July 10th. Of this number 295 terminated in recovery and 147 in death, a mortality of 33.3 per cent. To reach a true estimate of the value of the serum, however, not all of these cases should be included. A considerable number of patients were moribund at the time of the first injection, and lived but a few hours. Eliminating the cases, 49 in number, in which death occurred within less than twenty-four hours, or before a sufficient time had passed for the serum to manifest its activity, but including all others, no matter how late in the disease the first injection was made, there remain for analysis 393 cases. In this number it may be said that the serum was given a fair trial. The mortality of these 393 cases was exactly 25 per cent.

"Even more accurately showing the effects of the serum are the results when the cases are grouped according to the time of receiving the first injection. This is given in the reports of 328 cases:

Day Injected.	No. of Cases.	Deaths.	Mortality Percentage.
1st to 3d.............	121	18	14.9
4th to 7th............	100	22	22.0
After 7th.............	107	39	36.0

"In conclusion, although the number of cases treated is yet small, the evidence herewith submitted must, I think, convince any one who approaches the subject with a candid mind that the course of cerebrospinal meningitis and its termination are distinctly modified by the injection of the serum of Flexner and Jobling. In all places where it has been employed the results have been much the same, and those who have had most experience with its use have become its enthusiastic advocates. With improvement in the quality of the serum and a better understanding as to how it should be used, which time and further experience are certain to bring, we may confidently predict that the subsequent results will be even better than those given in this paper. From being one of the diseases most to be dreaded, both on account of its high mortality and its sequelæ, often worse than death, it seems probable that cerebrospinal meningitis will become, like diphtheria, one most certainly controlled by treatment."

A very extensive discussion followed the reading of Holt's paper. Prof. Kolle, of Berne (whose serum will be alluded to, p. 68), strongly advocates the serum treatment, which he characterizes as harmless. Dr. C. B. Ker (Edinburgh) said that he had treated 30 cases in the Edinburgh City Hospital with Flexner's serum. Of these, 17 recovered and 13 died, the mortality being thus 43 per cent., as against 73 per cent. for the whole outbreak of 138 cases, including those treated with that serum, and against 80.5 per cent. for the 108 cases not so treated. While these figures were not so good as those reported from other centers, they included no less than 3 patients who died within twelve hours of admission, and in some of the earlier cases it was probable that the serum was not sufficiently pushed. The most striking effects of the serum injections were, first, the tendency of the disease to terminate by crisis; secondly, its marked influence in checking relapses; and, thirdly, its success in young children. Among the latter the death rate for the epidemic was 81 per cent., while for serum-treated cases it was only 21 per cent. The serum was always injected into the spine, and an average dose for an adult was 30 to 45 c. c. This should be repeated daily till results are obtained.

Very interesting were the remarks by Dr. Gardner Robb, of Belfast, Ireland. He said: "The recent outbreak in Belfast commenced at the beginning of last year. From that time up to the end of last August, 275 cases had come under treatment in the fever hospitals of which I have charge. In these cases almost every form of treatment which had been suggested was tried. The case mortality was 72.3 per cent. Included in these 275 cases were 69 cases treated by *hypodermic* injections of various forms of antimeningitic serum (three different varieties were used—Kolle and Wassermann's, Ruppel's, Burroughs and Wellcome's), but the death rate in these was not better, being in them 74 per cent. At the beginning of last September I began the use of intraspinal injections of Flexner and Jobling's serum. I was so impressed with the results that I dropped all other forms of treatment, and since that time *every* case admitted to hospital has been treated in this way. No selection of cases has been made. In only one case was the serum not used, and that case died a few minutes after admission and before an injection could be given. The total number of cases thus treated was 90. The diagnosis in all these was confirmed in the bacteriological laboratory of Queen's College. Of these 90 cases, 27 died, giving a case mortality of 30 per cent.; so that in 275 cases treated by other means the death rate was 72.3 per cent., while in the 90 cases treated with Flexner and Jobling's antiserum it was 30 per cent. This alteration in the results obtained has been very striking, but not more striking than the change in the appearance of our cerebrospinal wards since this treatment was adopted. To any one having experience of this disease the picture of cases becoming chronic and dying slowly with progressive emaciation and increasing cerebral pressure, running on into weeks and months, must be very familiar. This class of case completely disappeared from our wards, and was no longer seen. Although there can be no question that the earlier this treatment is commenced the better the outlook, some of our most satisfactory results have been obtained in cases already "chronic" in type before being admitted to hospital. The method of using the serum has varied little with us. As soon as possible after admission, lumbar puncture is performed (under chloroform unless the patient is comatose), and as much cerebrospinal fluid as will is allowed to flow off, and 30 c. c. of serum injected into the canal. In a few, but only a few, a larger dose of 40 or 45 c. c. was given. In the more severe cases the injections were repeated daily for three or four days, and then, if still necessary, at longer intervals, until convalescence was established, the total amount of serum injected varying from 30 c. c. to 330 c. c. In some it was found that even when a large trocar was used pus was obtained so thick that it could not be got to flow. In such cases a few syringefuls of normal saline were injected and allowed to flow off again, and this repeated several times, so that the lower part of the canal was fairly thoroughly washed out, and the serum was then injected."

(b) *Other sera.* J. R. Currie and A. S. Macgregor[1] recount their experience with serotherapy in cerebrospinal meningitis in the City of Glasgow Fever Hospital between May, 1906, and May, 1908, in 105 cases, as compared with 225 cases not so treated. Four sera were used, Wassermann's, Ruppel's, Kolle's, and Burrough, Wellcome & Co.'s. Wassermann's serum was given to 20 patients, with 13 deaths; Ruppel's to 6, with 5 deaths; Kolle's to 20, with 14 deaths; and Burroughs, Wellcome & Co.'s to 56, with 34 deaths. Three patients, with 2 deaths, had more than one kind of serum administered.

Injection was both subcutaneously and intraspinally. Not more than 25 c.c. were injected at one time into the spinal canal. Intravenous injections were also given. Few cases had one injection only, and several had more than 10, one patient receiving 21. "Among the 105 serum-treated cases, with 68 deaths, the case mortality was 64.8 per cent. Among the 225 untreated cases, with 179 deaths, the case mortality was 79.5 per cent. Considered with reference to the number of cases involved and also with reference to the fact that many cases admitted *in extremis* during the acute phase of the illness did not receive serum treatment, the difference between these percentages is barely significant."

In France the serum treatment of epidemic cerebrospinal meningitis has not made much headway. Thus we find

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(1) The Lancet, Oct. 10, 1908.

F. Carles[1] reporting a case properly observed and diagnosed, and cured by repeated lumbar puncture. "Warm baths and lumbar puncture repeated until the cerebrospinal fluid was of normal appearance and of normal cytological composition were the only therapeutic means employed by us against this disease." He goes on to say: "We are more and more relying upon lumbar puncture as the means both of diagnosis and of therapy in our children's service."

A case of very grave cerebrospinal meningitis cured by intraspinal injections of antimeningococcic serum is reported by Descos and Vidal.[2] The patient, a robust young man of 19 years, was suddenly seized January 31 with a violent headache. In twenty-four hours he was comatose. On Feb. 2, 10 c.c. of Wassermann's serum was injected into the spinal canal, and 20 c.c. Feb. 4. The temperature fell after both injections and the general condition was improved. On Feb. 6, 10 c.c. of Dopter's serum were injected, and on Feb. 10, 10 c.c. in addition. Recovery followed rapidly, being complete by Feb. 27, on which date the patient was discharged.

Very interesting [and very like the case with Flexner's serum.—Ed.] was the behavior of the cerebrospinal fluid. At the first puncture, before there had been any treatment, the count showed 90 per cent. polynuclear leucocytes and 10 per cent. of mononuclears. The leucocytes showed numerous diplococci in their protoplasm, even ten or a dozen or more. Examination of the fluid Feb. 2 was not made, but the cerebrospinal fluid obtained Feb. 4 showed the polymorphonuclears reduced to 75 per cent., the mononuclears increased to 25 per cent., and about three-fourths of the diplococci were gone. More of extracellular diplococci were found. Examination of the fluid obtained Feb. 6 showed 20 per cent. polymorphonuclears, 80 per cent. mononuclear lymphocytes, and no diplococci at all. Only 15 to 20 leucocytes were now found per field. On Feb. 10 the cerebrospinal fluid was found to be perfectly limpid, without diplococci.

Brelet[3] says that in France the use of repeated lumbar puncture formerly advocated by Netter, the injections of

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(1) Jour. de méd. de Bordeaux, Jan. 3, 1909.
(2) Lyon Méd., April 18, 1909.
(3) Gaz. des Hôp., April 6, 1909.

collargol and electrargol, and all other methods are being abandoned for Flexner's serum, prepared by Dopter at the Pasteur Institute. His observations are precisely like those made by American authors. He called attention to the great reduction in mortality in America and England, and advocates its very early use in all cases, its efficiency depending upon the date of its administration. If in a suspected case a turbid fluid is withdrawn on lumbar puncture, an injection should be made then and there, so as not to lose time in laboratory examinations. As much cerebrospinal fluid as possible should be removed, and 20 to 30 c. c. of the serum, warmed to body temperature, introduced. Often the improvement is very immediate and striking. In general it may be said that French observers are finding in Flexner's serum all that its American enthusiasts have claimed for it.

The *technic* for intraspinal injections of antimeningococcic serum is given by C. Dopter[1], his directions being those previously worked out in America on Flexner's serum. Subcutaneous injections are ineffectual. About as much cerebrospinal fluid must be withdrawn as one wishes to inject of serum. Generally 20 to 35 c. c. should be injected, immediately after which the patient should have the pelvis elevated to aid the flow of the serum through the spinal canal. In this position he should remain from 6 to 8 hours.

TUBERCULOUS CEREBROSPINAL MENINGITIS.

A case of tuberculous cerebrospinal meningitis with secondary infection is reported by G. Paisseau and L. Tixier[2]. The patient, a girl of 8 years, entering the hospital as a case of appendicitis, showed on the contrary signs of cerebrospinal meningitis, her legs being drawn up, the pupils unequal, and the left eyeball showing internal strabismus. The neck was rigid, Kernig's sign was present, respiration was sighing, the belly retracted, the pulse irregular, the bladder distended. Lumbar puncture gave issue to 20 c. c. of cerebrospinal fluid showing a pure lymphocytosis, and a number of red blood corpuscles. Microscopically, the

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(1) Le Progrès Méd., April 21, 1909.
(2) Gaz. des Hôp., July 8, 1909.

fluid showed a few diplococci resembling those of the epidemic form of cerebrospinal fever. The cultures gave a diplococcus negative to Gram and in every way resembling the *diplococcus intracellularis meningitidis* of Weichselbaum. But since the germ grew rapidly in all sorts of media, being grown on some twenty kinds, the agglutination test was tried, and found negative. This diplococcus was therefore a pseudodiplococcus.

A second lumbar puncture was made the next day, and 15 c. c. withdrawn. The cell findings had markedly changed. From a pure lymphocytosis, the cerebrospinal fluid had changed its cell contents to the following formula: Polynuclears, 58 per cent.; lymphocytes, 30 per cent.; mononuclears of average dimensions, 12 per cent.; and there was still a good deal of blood. No tubercle bacilli could be found. An inoculation of a guinea-pig was made with some fluid obtained the next day, and one month later the animal died, showing numerous subperitoneal tubercles.

The child died within a week, and at the autopsy numerous tubercles were found in the cerebral meninges, especially in the fissure of Sylvius. In the mediastinum were some caseated glands, and the apex of the left lung showed a focus of tuberculosis.

INFLUENZAL MENINGITIS.

A case of influenzal meningitis from the Johns Hopkins Hospital is reported by B. A. Cohoe,[1] and the subject is thoroughly discussed. On admission the patient was found to be extremely restless and at times delirious. The neck was stiff and retracted, and Kernig's sign was present on both sides. The right eye showed a slight external strabismus. The pupils were small, but otherwise negative. Both discs were hyperemic, edematous, and slightly swollen. The arteries and veins were large, and the latter were quite tortuous. Lumbar puncture yielded a turbid fluid, under a good deal of pressure. The temperature ranged from 100° to 104° F., the pulse from 64 to 95 beats per minute. The leucocytes rose as high as 13,000 to the cubic millimeter, and showed a predominance of polymorphonuclears.

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(1) Amer. Jour. of Med. Sciences, January, 1909.

During the first four days there was retention of urine and catheterization was necessary.

The cerebrospinal fluid showed an organism which on pigeon-blood agar gave a luxuriant growth of dew-drop colonies, while subcultures on hemogoblin-free media yielded no growth. Morphologically it resembled a diplobacillus, and was non-motile and Gram-negative. The culture conformed biologically and morphologically with the bacillus of Pfeiffer. The same bacillus was obtained from a second and third puncture. Blood cultures were negative.

An historical review of cases of influenzal meningitis previously reported follows. The question of the portal of entry of the influenzal bacilli into the central nervous system is problematical. Since influenza is characterized by rhinitis, the nasopharynx is doubtless the point of entrance. The middle ear is a likely avenue of infection, due to the frequency of otitis media as a complication. Lastly, a general infection with the influenza bacillus is a possibility.

Symptomatology. The onset may be sudden without prodromal symptoms, beginning with a chill, high fever, and marked cephalic disturbance. When the base of the brain is first attacked, the clinical picture closely resembles the epidemic form of cerebrospinal fever. Usually the prodromal symptoms include those of the respiratory type of influenza, headache, rhinitis, bronchitis, general malaise, and fever. Gradually the meningeal symptoms predominate. The head is held retracted, and passive movement is painful. If the lesion is basal the head may be turned toward the right or left, and there may be facial paralysis. Kernig's sign is present as a rule. The ocular muscles are usually involved, although the eye symptoms are variable. The pupils may be unequal, and react poorly; the fundi are negative in the majority of cases. A general hyperesthesia may be present. The pulse rate in infants may be rapid, but bradycardia is not uncommon, a rate as low as 50 being observed in several cases. The reflexes are variable. A positive Babinski phenomenon has been found in most cases. The abdominal and epigastric reflexes may be absent. Mental symptoms are common. The *prognosis* is grave. Of 26 cases collected, a fatal issue resulted in all but 4. As to treatment, the indications are for those of meningitis.

A case of influenzal meningitis in an infant 4 days old is reported by D. J. Davis[1]. A purulent cerebrospinal meningitis was found at the portmortem examination. Clinical signs of meningitis were not prominent. Death occurred 4 days after birth, and the necropsy showed a purulent meningeal exudate with a pure growth of influenza bacilli. The atria of infection usually infected in these cases were normal. An acute enteritis existed. There was no evidence of influenzal infection in the mother or other members of the family.

MENINGITIS CARCINOMATOSA.

Meningitis carcinomatosa was reported in the Practical Medicine Series, Vol. X, 1908, p. 64, and two more cases are here presented by E. Stadelmann[2], both from the postmortem room. The first case occurred in a man 64 years of age, with stomach trouble, who had been confined to bed only 3 days. The last day there was difficulty of speech. The patient then became dull, showed marked stiffness in the neck muscles, the pulse was 60; there was no fever. Enlarged glands were found in the left supraclavicular fossa, and a tumor in the epigastrium. The knee-jerks were lively, the Babinski toe-phenomenon was bilaterally present, and there was double choked disc. Lumbar puncture gave an initial pressure of 220-230 mm. After 8 c. c. of fluid had been removed, the pressure fell to 140 mm. and the needle was withdrawn. The fluid was cloudy, showed lymphocytes and red blood corpuscles, and some round cells with a large nucleus. See Fig. 4. These cells could hardly be other than carcinoma cells. Hence the diagnosis of meningitis carcinomatosa. This diagnosis was confirmed by postmortem examination, a primary tumor being found in the stomach, with a beginning purulent meningitis and a less extensive carcinomatous meningitis in the region of the left cerebellar hemisphere. Moreover, the same cells were found in sections of the cerebral meninges.

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(1) Jour. Nerv. and Ment. Diseases, August, 1909.
(2) Berliner klin. Woch., Dec. 21, 1908.

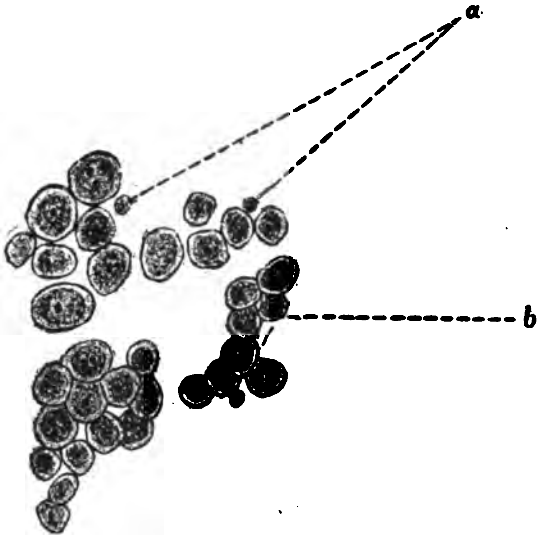

Fig 4.—a=Small lymphocytes; b=Endothelial (carcinomatous) cells. Leitz, Ocul. 2, Objective 7, Tube-length, 135.

A second case is mentioned in a man 33 years of age, with carcinoma of the stomach, the diagnosis of meningitis carcinomatosa being made by lumbar puncture. Besides these two cases, Stadelmann has found a record of three other cases of lumbar puncture in carcinomatous meningitis, but in these three cases no pathologic cells were found in the cerebrospinal fluid withdrawn.

TUMOR OF THE MENINGES.

Courmont, Savy, and Lacassagne[1] report a case of voluminous tumor of the meninges over the external surface of the right hemisphere, unsuspected during life, a diagnosis of progressive paralysis having been made. The case is remarkable in its symptomatology and merely goes to show how difficult at times is the diagnosis of cerebral neoplasm. The patient, 58 years of age, denying syphilis, showed progressive loss of memory, especially for recent events; speech

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(1) Lyon Méd., April 25, 1909.

was hesitating and mumbling; his writing showed numerous faults, was tremulous, with letters often out of line, transposed, or used for other letters. His legs were stiff, walking was difficult, with small steps, the reflexes were increased; and though the pupils reacted to light, there was myosis. In 4 months death followed, and at the postmortem examination the tumor previously mentioned was found. It had developed from the meninges, and not from the brain substance. The most noteworthy point is the disproportion between the symptoms and the size of the tumor. There was no limitation of motion anywhere on the patient's left side.

MENINGOENCEPHALITIS AND JACKSONIAN EPILEPSY.

Lannois and Durand[1] report a case of Jacksonian epilepsy in a young man of 26 years of age, who at 21 years of age had an otitis media, following sore throat. A year later he was suddenly seized with a severe convulsion, without aura, which felled him to the ground. He remained unconscious for 3 hours, but did not soil his clothing. The next two and a half years he remained well, until he was suddenly seized without an aura or loss of consciousness, by a convulsion involving only his right side. Perfectly conscious throughout, he remained stretched out on the ground 2 or 3 minutes, and got up without assistance. Thereafter the Jacksonian crises were frequent, and some were attended with loss of consciousness. After enduring these convulsions for 6 months he consulted the authors, who trepanned, and found over the left motor area a patch of meningoencephalitis resulting from a healed meningitis. It could not be removed, and as a result the operation was wholly unsuccessful in relieving the patient's attacks.

Such a cause of Jacksonian epilepsy is very rare, being the second only in the experience of the authors, the first case being in a child with meningitis following bronchopneumonia and measles. Needless to say this case also was not improved by operation, the brain tissue beneath the meninges being much affected.

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(1) Lyon Méd., Oct. 4, 1909.

CEREBRAL RHEUMATISM.

Cerebral rheumatism is mentioned by H. H. Hoppe[1], and a number of authors quoted, but no mention is made of Lépine's cases, last year summarized briefly in the Practical Medicine Series, (1908, Vol. X, p. 63). In modern pathology this disease seems scarcely to have a place, but nevertheless Hoppe reports a case in a man, 43 years of age, whose illness began with pain and swelling of the right hand, fever, and acid sweats. Various joints were affected. There was some delirium, and hallucinations of sight and hearing. The temperature was not specially high, until the cerebral symptoms appeared, then it rose rapidly, coma followed, with rapid pulse and respiration, and inequality of the pupils. In 10 hours the patient recovered consciousness, and in 3 or 4 days gradually returned to a normal mental state. A description of the disease follows, with a differential diagnosis from salicylic acid intoxication, delirium tremens, sunstroke, and non-purulent, hemorrhagic encephalitis. The prognosis is always very serious. The London Clinical Society gathered 107 cases [no reference by Hoppe as to the date of this report.—Ed.] of whom 57 died and 50 recovered.

TRAUMATIC SUBDURAL HEMATOMA.

A case of traumatic subdural hematoma trephined 3 months after injury is reported by G. H. Hume[2]. The case is mentioned merely to illustrate the obscurity which may mark the course of cases of subdural hemorrhage, and the excellent results of operative intervention.

"The patient, a man between 50 and 60 years of age, fell from his bicycle in the early morning of August 9, 1907, and received a cut on the back of the head on the left side. He lay unconscious for a few minutes and came to himself standing in the midst of a group of workmen who had come to his assistance. The wound was dressed by a surgeon later in the day. It healed readily and the ordinary habits of life and business were not interrupted. But from the

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(1) Klinisch-ther. Woch., Oct. 26, 1908.
(2) The Lancet, Sept. 19, 1908.

date of the accident he felt vaguely ill and suffered from a constant feeling of anxiety and depression. This feeling seems never to have left him, and in a written statement which he has furnished he gives so graphic an account of his condition to the time of his complete breakdown that I am tempted to transcribe the more salient parts of his story. After dwelling on his ever-present anxiety and trouble of mind, he says:

"'About the middle of September I had 3 weeks' leave (the remainder of my annual vacation), the first few days of which I spent with friends in the country. From there I went on a voyage to Hamburg, where I lived on the steamer in one of the harbors. The sea and fresh air did me good, but I still suffered from want of sleep and the ever-recurring feeling of worry and anxiety about anything I had to undertake. I confess to a constant urging towards suicide; it seemed so easy to drop overboard in the dark and end all my troubles. When I returned to work I began to suffer from pains in the left side of my head, particularly if suddenly startled or confronted with any difficulty in business, which at that time seemed mostly difficulties. My handwriting deteriorated, my signature became difficult. Towards the end of October I began to have great difficulty in shaving, owing to loss of power in my right hand; I also found it trying to walk up stairs and hills. I had very constant urgings to pass water and began to suffer from constipation, a very unusual thing with me. I finally broke down on Tuesday, October 29th, on which day I could hardly sign my name and had great difficulty in getting up the hill on my way home. After my collapse my memory is very confused and I have great difficulty in distinguishing between things that really happened and what must have been mere dreams. But I can remember something of most days except the 4 before November 14th, the day of the operation. These seem a blank, during which I must have suffered much from my head, but all this I have forgotten.'"

When seen November 1, the patient was drowsy and complained of headache. He had vomited suddenly several times. There was paresis of the right hand and leg. On November 7 all the symptoms had increased in gravity. He was more drowsy and breathed stertorously when asleep.

"When roused he had a pained and puzzled expression, and could not find or form the words he evidently wanted. There was right facial paralysis in addition to more marked affection of the arm and leg. Motions and urine had on several occasions been passed in bed. The knee-jerks were absent and there was no ankle-clonus or plantar response. The optic discs were congested, with blurred edges. The temperature was slightly raised and the pulse was of moderate rate, not characteristic of cerebral pressure. He could speak but not intelligently. Evidently irritated by the chimes of the neighboring cathedral, he said repeatedly, 'I want it disconnected,' but could not explain what 'it' was. He could not recognize objects handed to him or in the room—at any rate he could not name them. He was able to read aloud from a book but could not repeat consecutively more than three or four words dictated to him. He could not carry out any commands spoken or written.

"It will be seen that the signs of pressure widely affecting the left hemisphere were unequivocal, but it was difficult to arrive at any decided opinion as to the precise agent causing the pressure. The long period that had elapsed since the accident, the readiness with which the wound had healed, and the absence of any adhesion of the cicatrix to the bone, or other indication of the bone having suffered damage, and the freedom of the patient from any sign of septic infection, all weighed against the presence of pus. As there had been no sign of compression closely following the injury, and as the case only reached a somewhat abrupt climax 3 months afterwards, it was not easy to interpret the condition as arising from a hemorrhage the direct consequence of the accident."

Operation was determined upon, and the skull opened over the posterior parietal region, the cicatrix of the old wound being included in the upper and posterior angle of the flap. The underlying bone showed no trace of injury, but beneath the bluish dura were found 5 or 6 ounces of a brownish fluid, and a large cavity extending downwards and forwards and loosely filled with clotted blood. The surface of the depressed hemisphere was not injured. Improvement followed upon the operation very rapidly, the mental faculties were restored, and the use of the arm and leg, and the power of speech regained.

SUBARACHNOID HEMORRHAGE.

Cases of subarachnoid hemorrhage are said not to be excessively rare, and to be curable, by Braillon[1], who reports a case in a high school boy, 16 years of age. Their cause is (1) trauma with or without fracture of the skull, (2) rupture of blood-vessels, attributable to atheroma, syphilis or sunstroke (Dopter), (3) hemophilia, and (4) unknown. To this latter category the case reported belongs. The boy rose in the morning as usual, but felt a numbness in the whole right side of the body. While dressing he completely lost consciousness, and remained in that state all day. The next morning he awakened, but tired rapidly when questioned, and was paretic over his whole right side, including the facial and hypoglossal nerve-supply. At the same time the neck was stiff, Kernig's sign was present on both sides, there was severe headache, especially in the left frontoparietal region, constipation was marked, the belly contracted, the temperature slightly elevated, the pulse lowered to 46 beats per minute, and the skin showed marked vasomotor irritability. Lumbar puncture was done, and unexpectedly showed a hemorrhagic cerebrospinal fluid. At first a tuberculous meningitis was feared, on account of these findings. On the other hand the pupillary reflexes were normal, the pupils equal and slightly contracted, the tendon reflexes slightly diminished, the cutaneous reflexes normal, and Babinski's toe-sign was absent on both sides.

After the lumbar puncture the headache began to fade away, the pulse rose to 60, the temperature fell to normal, Kernig's sign completely disappeared, the hemiparesis improved, there was no facial assymetry, and altogether the boy was better. Cultures taken of the cerebrospinal fluid were negative, and two guinea-pigs, inoculated with it, were found perfectly healthy at the end of two months. There was no tuberculosis, and no evidence of either hereditary or acquired syphilis could be found. The boy steadily improved, and about six weeks after the attack was wholly recovered. The author concludes that there was a rupture of an arachnoidian arteriole somewhere in the left Sylvian region, and that the escape of the corpuscles beneath the arachnoid caused sufficient meningeal irritation to constitute an aseptic meningitis. The cause of the rupture is unknown.

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(1) Gaz. des Hôp., May 4, 1909.

CEREBRAL ANEURYSM.

Aneurysms of cerebral vessels are discussed by John R. Bradford.[1] In order of frequency the vessels involved are the middle cerebral, the basilar, the internal carotid, and the anterior communicating. Though aneurysms have been described elsewhere they are too infrequent to merit special mention. Characteristic of cerebral aneurysms is the fact that they are saccular and very thin-walled. The size varies enormously, from a pin's head to a walnut, the average being the size of a pea. Aneurysms may occur at any time of life, but most of them occur before the fortieth year. They have been known to occur in childhood. Clinically, cases of cerebral aneurysm may be classified as follows: Group I, cases that produce no symptoms at any time, a considerable proportion of all cases. Group II, cases that present simply the symptoms of cerebral hemorrhage, generally terminating rapidly in death. Group III, cases that present symptoms more or less characteristic of cerebral tumor, from which the differential diagnosis is difficult to make. Group IV, cases presenting irregular cerebral symptoms, a class in which there is a definite clinical course, and in which a diagnosis may be hazarded. These cases run in two stages. In the first stage obscure and sometimes seemingly trivial symptoms develop, and then after a short duration of a few days or weeks, the second stage of illness develops with hemorrhage and death.

A case of *aneurysm of the anterior cerebral artery* is reported by A. S. MacNalty.[2] The patient, 28 years of age, was suddenly attacked with severe headache April 27, 1908, while at work. The headache was across the top of the head and soon after its onset the patient vomited. He went home, and to bed. The time occupied in walking home was about eight minutes. When in bed he vomited again and immediately lost consciousness. He remained insensible for 20 minutes, becoming quite rigid; he had no twitchings and no incontinence of urine or feces during the attack.

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(1) The Lancet, Sept. 5, 1908.
(2) The Lancet, Dec. 5, 1908.

For the next three days he vomited several times a day irrespective of eating. After April 30 he had no further sickness, except an intense occipital headache, which disappeared in three days. On May 1 he took a short walk, but the effort fatigued him and he went back to bed. Meanwhile the sight of the right eye had been failing and he began to see double. By May 7 he was almost blind, and there was increasing weakness in all four extremities. Examined May 7, he was found to be somewhat drowsy and his speech was slurred in character. "The pupils were equal and they reacted to accommodation. The left pupil reacted to light; the right pupil did not react to direct, but did to indirect, stimulation. The patient was blind in the right eye. Visual perception was normal in the left eye. There was no nystagmus. With regard to the cranial nerves there was slight weakness of the masseter on the left side. Both external recti were paralyzed, the left more than the right. There were marked ptosis of both eyelids, weakness of the left orbicularis palpebrarum, and obvious facial weakness on the left side. Hearing appeared to be diminished on both sides. The other cranial nerves were normal. Ophthalmoscopic examination revealed marked optic neuritis in the left eye with hemorrhages in the disc. The right fundus was normal. The motor power was weak in the arms and the legs. There was no anesthesia, analgesia, or atopognosis. The patient was unable to touch accurately the tip of the nose with his forefinger and could not feed himself. The superficial reflexes were normal, as were also the deep reflexes of the arm and the wrist-jerks. The knee-jerks were absent, a condition which persisted throughout the illness. No murmur was heard on auscultation of the head. The diagnosis was that of cerebral aneurysm.

"There is nothing of note to record until May 16, when the patient was found to have atopognosis (being unable to localize accurately the position of things touched) and astereognosis. Both superior oblique muscles of the eyeball appeared to be paralyzed. Occasionally the patient showed athetoid movements (tabetic type) in the hands. His other physical signs were unchanged, but his general condition had much improved and he took his food well. On the 18th he appeared to have regained more power in his limbs. On the 20th the end came with dramatic suddenness. When examined at 11 A. M. the patient was quite rational and announced that he was feeling much better. The temperature was 96°, the pulse was 84, and the respirations were 20. At 11:30 A. M. he suddenly called out. On coming to the bedside he was found to be unconscious; his breathing was stertorous and his face was deeply cyanosed. There was some twitching of the left side of the face and of the right upper eyelid. The knee-jerks were unaltered and there was no rigidity of the body or the limbs. The patient had a stool just previous to the attack. Oxygen was administered and five minims of solution of strychnin were given hypodermically. Death ensued within ten minutes from the onset of the seizure. On auscultation the heart was heard to beat feebly for a short time after the cessation of respiration."

The postmortem examination disclosed a blood clot at the base of the brain "extending from the optic chiasma over the floor of the third ventricle to the pons and medulla and extending between the cerebellum and the cerebrum. There was blood in both lateral ventricles. The hemorrhage extended downwards along the cord as far as the third cervical root. On dissection a thin-walled saccular aneurysm was found in the left anterior cerebral artery occurring in its course just after it had given off the anterior communicating branch. The aneurysm was of about the size of a small cherry; it had exercised so much pressure upon the brain substance that it could be seen bulging into one end of the left lateral ventricle, the small mass of blood clots that it contained showing up through the thin wall of the aneurysmal sac. In this region it had caused a certain amount of disorganization of the adjacent brain substance and the inner wall of the left ventricle showed much ploughing-up. On the other hand, it had crossed the middle line to exert pressure on the right optic nerve. All round the optic nerves the brain showed a considerable amount of brown staining. The blood from the aneurysm took the path usually pursued by it in aneurysms rupturing at the base of the brain. Instead of tearing through the pia mater the blood was extravasated between the membrane and the brain substance; it tracked round the optic chiasma and pons to the side of the medulla and thence poured by way of the iter into the ventricles.

An *aneurysm* of the left anterior cerebral artery with rupture, *simulating brain tumor,* is reported by J. A. Booth.[1] The patient, a man thirty years of age, when seen in 1907 had had in January, 1906, a convulsion with loss of consciousness, followed by deep stupor. Similar seizures at frequent intervals followed, without aura, or localized spasm of muscles. There was rigidity of the neck, marked Kernig's sign, slight ataxia in the right hand, equal and exaggerated knee-jerks, no sensory disturbance or loss of any of the special senses, no paralysis of single or associative ocular movements, no ptosis, pupillary inequality or perverted reflexes. Yet opthalmoscopic examination showed a double optic neuritis with choking of the discs. A diagnosis of brain tumor, complicated by a basilar meningitis, either tubercular or syphilitic, was made. Lumbar puncture disclosed no evidence of tuberculosis, and antisyphilitic treatment was therefore commenced. Decided improvement followed both as to headache and mental condition. On July 3, however, just 30 days after the above examination was made, the patient was seized with a severe convulsion followed by deep coma and death.

The postmortem examination showed a large blood-clot the size of a small peach, containing within it pieces of necrotic brain tissue, in the left frontal region. It involved the first, second and third frontal gyri. On the base of the brain, under the pons, cerebellar crura, and interpeduncular space, was a recent subpial clot extending backward behind the medulla towards the foramen of Magendie. The source of the blood was a ruptured fusiform aneurysm of a primary branch of the left anterior cerebral artery.

CEREBRAL BLASTOMYCOSIS.

A case of blastomycotic lesions of the brain is described by E. R. Le Count.[2] A case which was previously reported by Le Count and Meyers[3] involved the entire outer half of the right hemisphere of the cerebellum. It was an irregularly shaped mass the size of a pigeon's egg, roughly resembling the so-called "solitary tubercle" of tubercu-

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(1) Jour. Nerv. and Ment. Dis., September, 1909.
(2) Jour. Nerv. and Ment. Dis., March, 1909.
(3) Jour. of Inf. Dis., December, 1907, 4, 187.

losis, apparently made up of agglomerated smaller nodules, some of which had a slightly softened necrotic center. Further examination of the cerebrum, after nothing was found in making frontal sections 2 cm. apart, resulted in the discovery of a second lesion, 8 by 10 mm., in the internal capsule.

The second case showed numerous small and widely scattered areas of blastomyces. "The first frontal sections of the brain, numbering seventeen and dividing the brain into eighteen segments, revealed six lesions, four in the cerebrum and two in the cerebellum, located as follows: One 2 cm. behind the anterior end of the left hemisphere, circular in shape, 2 mm. in diameter and 2 mm. beneath the gray substance of the cortex; a second in the gyrus fornicatus of the opposite hemisphere, slightly smaller just above the corpus callosum, and 7.2 cm. from the anterior end of the hemisphere; the third and fourth, about equal in size, located at the same distance from the anterior end of the right hemisphere—8.7 cm.—one in the optic thalamus and one just beneath the gray substance of the cortex; and the fifth and sixth lesions in the cerebellum.

"After these six separate lesions were found by the seventeen frontal sections mentioned, the entire brain was further subdivided by twenty-four more frontal sections, some of the thicker segments resulting from the first sectioning being divided twice. In consequence of the added surfaces exposed eight additional foci were found. For the most part these were all small and quite superficial, located at the bottom of the sulci, in the gray cortex or at the juncture of the cortex and white substance, and all but two in the right hemisphere. (See plate I.)

"In their dissemination, color and occasional location in the pia these more minute blastomycotic nodules have some points of similarity to the pial miliary lesions of tuberculous meningitis. With certain features of resemblance between the lesions of blastomycosis of the central nervous system and both the large 'solitary tubercle' and the minute discrete miliary tubercle, it will not be surprising if in future postmortem examinations a diffuse fibrino-purulent blastomycotic meningitis is observed in instances of systemic infection. Such a prediction seems warranted for the additional reason that the process results in a liquefaction necrosis not infrequently, the subcutaneous nodules for example, developing into abscesses quite regularly."

CEREBRAL PARASITISM.

A very peculiar case of disease of the brain caused by the Distomum Westermanni, presenting the symptom-complex of brain tumor is reported very elaborately by T. Tsunoda.[1] This parasite is a common one in Japan, being found chiefly in the lungs in the center of a cyst, and giving rise to a peculiar spitting up of blood. It is also found in the orbits, the eyelids, and in the testicles. Very interesting is its involvement of the brain. The first case, with symptoms of epilepsy, was reported by Otani in 1887-88; later Inouye reported 11 cases of distomum disease of the lung with brain symptoms. Other later writers with resumés of their cases are mentioned fairly completely, and postmortem findings by Otani, Katsurada, Taniguchi, and Karasawa are mentioned as the only four cases in the literature.

The present case was in a boy, 16 years of age, and began with violent headache, vertigo, a chill, and fever. The symptoms all gradually subsided, but headache and vertigo again returned and were severe. About four months later there suddenly appeared paraplegia with involuntary passage of urine. Later convulsions, first clonic, then tonic, swept over the whole body, and he lost consciousness. Subsequently there was vomiting, and more convulsions followed. Later a slight ptosis developed, and the right pupil was enlarged. Death followed a month later.

The autopsy showed adhesions between dura and pia, flattening of the gyri of the right hemisphere, which was larger than the left, and a cyst, the size of a dove's egg, fluctuating, in the anterior end of the right temporal lobe. This had caused a widening of the inferior cornu of the lateral ventricle. When the hemisphere was opened it was found to contain several irregular cavities from softening, and one large one, the size of a pigeon's egg, in the interior of the white matter beneath the frontal and parietal lobes. The cyst-wall was hard, and reddish brown. Other cysts were found in the optic thalamus, the lenticular nucleus, and the internal capsule. Not only was the lateral ventricle

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(1) Wiener med. Woch., Nov. 21 and 28, 1908.

dilated, but so also were the third and fourth ventricles. The left hemisphere was intact. A very careful description of the cysts and eggs follows. In summarizing the symptoms the statement is made that the essential one is the convulsions. Distomum seems to cause "a sort of cortical epilepsy."

CEREBRAL SYPHILIS.

Pathology. The pathology of syphilis of the nervous system in the light of modern research formed the title of F. W. Mott's Morison Lectures, delivered in Edinburgh Jan. 25, 27, and 29 of this year.[1] The specific organism may be definitely accepted as the *Spirochaeta pallida,* whether it be protozoon or bacterium. Inoculated in the chimpanzee it reproduces with absolute certainty the human symptomatology. The microbiology of the organism is carefully set forth, and a table of differences between the spirochaeta pallida and other kinds of Spirochaetæ is presented. The organisms after local development at the point of inoculation in man soon reach the nearest lymphatic glands, where they set up an adenitis; these changes may be biological, provoked by the organism for its perpetuation, and not in the nature of a defence on the part of the glands. As a rule the Spirochaete prevails and streams through the lymphatics, causing a polyadenitis. It is quite probable that from the thoracic duct a general infection of the blood stream takes place, with the development of the secondary eruption (roseola). Quite early in the disease, even before the primary sore is healed, symptoms pointing to meningitis may occur. Indeed, in some cases it is quite probable that the meninges are infected at the time of the rash, but that symptoms of this infection are overlooked.

The *Spirochaeta pallida* is probably a protozoon of the trypanosome group. Of interest is the fact that the clinical course of syphilis is unlike that of a bacterial infection. Furthermore the histological lesions of the nervous tissue in chronic trypanosome lesions resemble syphilitic lesions, thus indicating their relationship.

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(1) British Med. Jour., Feb. 20 and 27, 1909.

"Finally, the therapeutic agents, mercury and arsenic in the many forms employed, are specific for both trypanosome and spirochaete affections. They are not of much use for bacterial infections. Mercury, particularly in the form of inunction, is especially valuable; and this may be owing to the fact that it prevents the pullulation of the spirochaetes on the surface of the body, including the mucous orifices, a habitat which these organisms have found particularly favorable for perpetuation of the species by transmission to another individual.

"I have pointed out that, practically, the morbid tissue changes in syphilis are similar, whether the lesion be the primary sore or a gumma twenty years later; moreover, it is difficult to understand how the spirochaete, seeing that it has hardly ever been found in tertiary lesions, can produce the same specific cell hyperplasia so long after the primary infection. The following hypotheses may be put forward to explain the phenomenon of a gumma appearing spontaneously in the central nervous system long after the primary sore and apparent cure of the disease:

"1. The spirochaete, or some modified form of it, has remained latent in the tissues at the seat of the lesion, and, for some reason, inherent or otherwise, the resistance of the tissues at that particular spot has become lowered, and the organism has exerted again its specific activity—possibly in some not yet discovered intracellular form.

"2. The specific organism has remained latent in some other tissue—for example, the marrow of bone, the spleen or glands—and, escaping into the blood or lymph circulation, has, like a new growth, engendered a metastasis, which has developed and increased, producing a hyperplasia of the fixed tissue cell elements, conjunctival and endothelial.

"3. There may be varieties of specific spirochaetes, one of which may have an elective affinity for the central nervous system, as we know the *Trypanosoma gambiense* has. It is difficult to differentiate this trypanosome from other forms by morphological appearances; how much more difficult would it be to differentiate varieties of *Spirochaeta pallida?*

"4. The invasion of the body by the spirochaetes has altered the blood and lymph bio-chemically, so that the tissue reactions to all causes which would lead to injury may take on the specific character."

Serum Diagnosis. The serum diagnosis of syphilis by the Wassermann method[1] depends upon a reaction known as deviation of the complement discovered by Bordet in 1901, and independently by Gengou about the same time.

"We require first to immunize an animal against the blood of some other animal; for this purpose the blood corpuscles of a sheep are injected into the circulation of a rabbit. The blood serum of the rabbit is thus made hemolytic to the corpuscles of the sheep by virtue of an immune body *plus* the normal complement or cytase. The latter can be removed by heating at 56° C. for thirty minutes without destroying the former. We have thus the immune body, which by itself will not dissolve the washed corpuscle of the sheep. If, however, we add the normal serum of a guinea-pig, the amboceptor or immune body links up the complement or cytase and the corpuscles are dissolved.

"The second part of the experiment is the deviation of the complement, or its neutralization, so that hemolysis no longer takes place when the serum of the guinea-pig is added to the immune body and the washed sheep's corpuscles. This is effected by the presence of both antigen and antibody in the fluid to be examined. The serum, or cerebrospinal fluid, to be examined is mixed in varying dilutions with a watery solution of the liver of a syphilitic fetus, which will contain the antigen (lipoid). A small amount of the serum of a guinea-pig is then added, and the total volume made up to 2 c.c. with saline solution. The series of tubes containing these mixed solutions are placed in an incubator at 37° C. for one hour, and then the sensitized blood corpuscles are added. The mixtures are again placed in the incubator for two hours at 37° C., then taken out and put on ice overnight. The next morning the amount of hemolysis in each tube is estimated. If, on the one hand, antigen and antibody have been present they have united with the complement, and no solution of corpuscles will have taken place because the complement is fixed; if, on the other hand, the immune body (antibody) was not present then the complement (cytase) has remained free to act upon the sensitized corpuscles and

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(1) F. W. Mott, British Med. Jour., Feb. 20 and 27, 1909.

lead to their solution. A control experiment, using a normal serum, or cerebrospinal fluid, namely, one which contains no antibody, must be used at the same time."

As to the value of the reaction it may be said that Plaut obtained a positive result in 94 out of 95 cases of general paralysis with the cerebrospinal fluid. In 70 to 80% of the cases of tabes the result was a positive reaction. Of 46 cases of general paralysis examined under Mott's directions by Candler and Smith, 41 were positive or 89.1%. Two cases of tabes dorsalis failed to give the reaction, and a negative reaction was also obtained in a case of syphilitic meningitis.

Mott calls attention, however, to the fact that many doubts have been cast upon the interpretation of the facts whether the reaction is in any way due to syphilitic antibodies. Thus Levaditi and Marie demonstrated that normal liver can replace the syphilitic liver in the preparation of the antigen. Indeed, "The sero-reaction can be obtained with bile salts and lecithin, or with soap (Sachs and Altmann), cholesterin and vaselin (Fleischmann), although more feebly. The sero-reaction of syphilis and of general paralysis is the same, and is not due to the intervention of antibody or syphilitic antigen in the usual sense of the word, and has no relation with the *Spirochaeta pallida.* * * * I have also found that a cerebrospinal fluid which gives a positive Wassermann reaction, after removal of the protein content by precipitation with alcohol fails to give the reaction.

"*Summary.*—The original method of Wassermann is the most complicated, but is regarded by the majority of investigators as the most specific and reliable. Whatever may be the explanation of the facts, all the evidence goes to prove: (1) That these methods in the hands of competent observers afford a valuable means of diagnosis, and are especially useful when applied to the cerebrospinal fluid for the determination of the existence or not of general paralysis. (2) That similar substances, whether antibodies or not, occur in the serum of syphilitic and parasyphilitic persons in such quantities as are not found in the serum of normal persons or in the serums of people suffering with other diseases. (3) That similar substances are found in the cerebrospinal fluid of tabetics and general paralytics, and the amount of those substances which cause a deviation of the complement or a precipitation is in proportion to the activity and length of duration of the disease; that these substances are of tissue origin, or arise from tissue destruction caused in some way by the action present or past of the syphilitic virus. (4) It is probable that the syphilitic virus excites an increased unloosening of complex lipoid substances containing lecithin and cholesterin, etc., from the red corpuscles and cells of the body. (5) That this prevails through life, and in certain cases of syphilitic infection—namely, general paralysis and tabes—the central nervous system, which under ordinary circumstances is protected against the loss of its lipoid substances, takes part in the process, and this is manifested by the presence of lipoid and globulin in the cerebrospinal fluid which acts as the antibody in the reaction. This lipoid complex, as well as globulin, increases in amount as the process of neuronic decay proceeds."

Diagnosis. The diagnosis of syphilis of the nervous system was again considered by F. W. Mott in a later paper.[1] It is well to bear in mind that in such a widespread disease as syphilis *coincidence* as well as *cause* is of frequent occurrence, and in a diagnosis coincidence and cause must be differentiated. "The first point to decide is whether the nervous symptoms from which the patient is suffering are the direct result of the syphilitic virus upon the membranes, the blood-vessels and their sheaths, with secondary degeneration of the nervous elements (for example, (1) Gummatous meningitis—cerebral, cerebrospinal, spinal; (2) localized gummata, single or multiple, obliterative arteritis); or are the symptoms those of tabes or general paralysis? It is not always easy to decide, but it is well to bear in mind that the shorter the period which has elapsed between the primary infection and the onset of the symptoms the more likely it is to be due to the action of the syphilitic virus; it is very unusual for a generalized syphilitic meningitis to commence after the lapse of five years from primary infection; quite one-half of the cases manifest symptoms within the first eighteen months, in some the symptoms are obvious with the appearance of the roseolar rash, and a few cases have occurred when the primary sore was yet unhealed. A very different order of things to tabes dorsalis and general paralysis, for in these diseases it is very rarely that symptoms occur in less than four years after infection, and the average time is ten years."

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(1) British Med. Jour., June 12, 1909.

Very necessary is the early diagnosis of *diffuse cerebral syphilis,* for it is amenable to treatment; whereas general paresis with which it is confused is hopelessly progressive; and many cases are admitted to asylums and are more often than not diagnosed as general paralytics, being accordingly not treated with mercury, and accordingly not helped. Mott goes on to say:

"From a clinical point of view syphilitic dementia is accompanied by numerous mental and bodily symptoms. The mental symptoms comprise states of excitement and depression more sudden and accentuated than general paralysis; there are disturbances of consciousness in the form of somnolent, drowsy, semi-comatose conditions from which the patients emerge, again to relapse, and such remissions are especially significant. In the intervals there is not the same intellectual and affective deficiencies as in general paresis, nor does the same approbativeness, moral optimism, suggestibility, mobility, and automatism obtain as in general paralysis. The accompanying bodily troubles are coarse and obtrusive, and due to focal disseminated lesions in the brain and spinal cord, causing apoplectic and epileptiform seizures, followed by aphasia, paraphasia, apraxias, hemiplegias, monoplegias, paraplegias, triplegias due to affection of cortical centers and their projection systems; or paralyses and sensory troubles from affection of cranial nerves and their nuclei of origin arise. The clinical picture may be slow in its evolution; it is irregular, variable, and proceeds by fits and starts, with prolonged and sometimes indefinite periods of arrest. It is often a stationary dementia, and under the influence of mercurial treatment it may undergo regression.

"The best criterion lies in the mode of evolution of the dementia and paralysis. The evolution of general paralysis is slow, insidious, and progressive towards marasmus and death. Syphilitic dementia, especially if it be treated, remains stationary or tends to regression, ending in a state of acquired imbecility, of which the history affords the clue. In my experience dementia from congenital syphilis is much less frequently met with than juvenile general paralytic dementia."

The methods employed by Mott are as follows: (1) The Wassermann-Neisser-Brück method, (2) the Noguchi method, and cytodiagnosis. Briefly the Noguchi method "consists in boiling two parts of the cerebrospinal fluid with five parts of a 10 per cent. solution of butyric acid in saline solution for a few seconds and then adding one part of a normal sodium hydrate solution and again boiling briefly. A flocculent or granular precipitate is obtained on standing in parasyphilitic affections, due to the presence of a globulin. The test is useful in distinguishing general paralysis from other forms of insanity not associated with meningo-encephalitis."

As to cytodiagnosis, the number of leucocytes may be approximately estimated by the following methods: 1. A drop of the fluid which has been previously well shaken is placed in the chamber of a Thoma-Zeiss hemocytometer and the cells allowed to settle. The cells are then counted in the usual way. 2. The cerebrospinal fluid is centrifuged and the supernatant fluid poured off. The residue is removed by a capillary pipette, blown on to a slide and allowed to dry. This is then stained. The count will show with a magnification of 400 diameters more than two or three lymphocytes per field, whereas a normal cerebrospinal fluid so treated will not show so many.

Max Nonne[1] in his address before the Section on Nervous Diseases of the American Medical Association at Atlantic City, June, 1909, took up this same subject of the clinical diagnosis of the syphilogenous diseases of the central nervous system. This address was really the extension of a report of a similar nature made at the second annual meeting of the German Society of Nerve Specialists at Heidelberg, October, 1908.[2]

Tabes Dorsalis. Within the past six months Nonne examined 47 cases of tabes dorsalis for four reactions, that is:

1. Pleocytosis.
2. Increase of globulin (Phase I).

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(1) Jour. Am. Med. Assoc., July 24, 1909.
(2) Deutsche Zeitsch. f. Nervenheilkunde, Vol. 36, 1909, p. 38.

3 and 4. Reaction of deviation of complement in the blood serum and spinal fluid after Wassermann. Of the 47 cases, 19 were cases of rudimentary tabes, presenting merely combinations of single symptoms which occur in incipient cases, such as pupil anomalies, absence of tendon reflexes, gastric crises, etc. In these forty-seven cases a distinct lymphocytosis was missing only seven times. Four of these seven cases were cases of tabes imperfecta, two were pronounced cases, and one was an incomplete case grafted on a hereditary basis. These data show, with Nonne's previously reported 101 cases, and with cases in the literature, that pleocytosis is one of the most regular signs of tabes. It occurs just as regularly in the imperfect as in the perfect cases.

The reaction Phase I (increase of globulin) was missing only seven times, and in the same seven cases. In conformity with his former experiences, this material also showed that reaction Phase I is equivalent to the pleocytosis reaction.

During the last six months Nonne examined 17 cases of general paralysis for pleocytosis and Phase I. Only in a single case did he fail to find both reactions, and this was one of juvenile hereditary paralysis. In this disease, too, then, the reactions are equivalent, whether for rudimentary or advanced cases.

Of 18 cases of the pure forms of cerebral and cerebrospinal syphilis, examined since September, 1908, 15 showed both reactions, pleocytosis and Phase I, and in 3 both reactions were missing.

As to the complement fixation reaction which has been applied by Wassermann, Neisser, Brück and Stern for diagnostic purposes in syphilis, it may be accepted that this reaction is not specific for syphilis, in the sense in which it was held to be by the first classic authors. Thus the Hamburg scarlet-fever findings have been confirmed by others. On the one hand, the syphilitic antigen may be replaced by non-syphilitic organ extracts and certain chemical substances, and, on the other hand, the inhibition or restraint of hemolysis which results from "fixation of the complement" may also take place in diseases which are not syphilogenous, but are either certainly or presumably traceable to protozoa or bacilli. The reaction has also been found to be positive in leprosy and malaria.

Holle, so long the collaborator of Wassermann, has declared as follows: "The deviation of complement reaction is not specific to syphilis; it is, however, characteristic of it. Hecht, of Prague, has stated the same, and I can only associate myself with these, as my later remarks will show. I say, with Lapowsky: The reaction is not specific in the bacteriologic sense, but is clinically characteristic of syphilis."

Nonne's summary follows: "Paresis gives almost invariably all four positive reactions. Tabes shows almost invariably pleocytosis, Phase I and Wassermann reaction only in the blood; the same is true of the different forms of cerebral and cerebrospinal syphilis. For the differential diagnosis between cerebrospinal syphilis and cerebral tumor without a syphilitic history, the Wassermann reaction is of extreme value. Pleocytosis and Phase I play no decisive part in the differential diagnosis between cerebrospinal syphilis and cerebral tumor. The occurrence of the Wassermann reaction in the blood in presumable idiopathic epilepsy should give rise to grave doubts against the diagnosis and should render it extremely likely that the condition is a syphilogenous one. The same holds good for pleocytosis and Phase I." Pseudotabes alcoholica never shows the four reactions.

Symptomatology. A case well illustrating the occasional bizarre collection of symptoms occurring in cerebral syphilis is reported by Burton Chance, under the caption: "Oculomotor paralysis accompanied by facial palsy, neuroparalytic keratitis, and hemiplegia."[1] The patient, a negro woman thirty years of age, complained of inflammation in her left eye for about six weeks, when suddenly a complete ptosis supervened. There was also facial palsy on the left side, and when the lid was raised the globe rotated outward. It was deeply injected, and the corneal epithelium was macerated and steamy, staining deeply with fluorescin solution. Rapid improvement in the eyeball followed the administration of potassic iodid, but the ptosis

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(1) Am. Jour. Med. Sciences, August, 1909.

and facial paralysis were not helped. Indeed after a time anesthesia showed in the territory supplied by the superior and inferior maxillary divisions of the fifth nerve, severe nocturnal headaches began, and she was seized with a *left*-sided hemiplegia and hemianesthesia. At this time W. G. Spiller saw the patient and concurred in the diagnosis. Mercurial inunctions were at once started, followed in three or four weeks by marked improvement, the hemiplegia having largely disappeared and the facial paralysis being much lessened in intensity. Improvement steadily continued, so that at the present time, four years after the symptoms above reported appeared, she has little left except a diplopia on attempting to look up or down, a partial ptosis, and paresis of the two oblique and the superior and inferior recti muscles. The facial movements were all regained, but the skin on the left side supplied by the two lower divisions of the fifth nerve is still anesthetic. There is no hemiplegia. Very unusual in the case is the occurrence of left hemiplegia with left oculomotor paralysis. It is probable that the hemiplegia was caused by vascular disturbance in the right side of the brain, while the oculomotor palsy arose through a disturbance at the base on the left side.

Epilepsia Partialis Continua in cerebral syphilis is reported by W. G. Spiller and E. Martin[1], the case being operated upon. This is a form of epilepsy described by Russian writers, little known in this country. The name epilepsia partialis continua, was first employed by Koshewnikow, who reported 4 cases in 1894. Spiller's case, a negro, 22 years of age, with a clear history of syphilitic infection, had his first convulsion in July, 1908. "The fingers of the right hand twitched at first slightly but later more severely, and the convulsion confined to these members lasted about twenty minutes and left a paralysis of the hand persisting about fifteen minutes. He had another convulsive attack confined to the right hand in about three weeks. The whole right upper limb became implicated in the third attack, and this attack was followed by unconsciousness. There seems to have been involvement of the face and right lower limb also in the fourth attack. After

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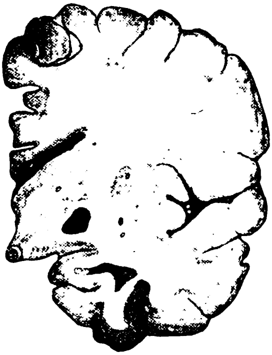
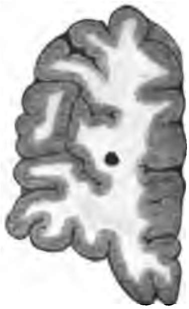
(1) Jour. Am. Med. Assoc., June 12, 1909.



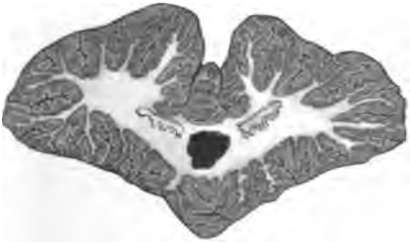
a.

Fig. a.—Posterior surface of a section through the right hemisphere illustrating the location of a nodule in the internal capsule.



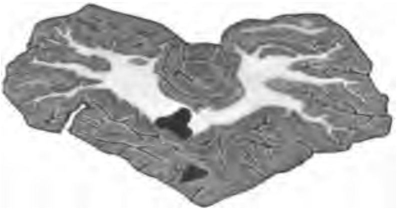
b.

Fig. b.—Posterior surface of a vertical segment through the left hemisphere 2 cm. from the anterior pole. Lesion in the white substance of the left hemisphere.



c.

Fig. c.—Large nodule in the posterior and central portion of the cerebellum. Posterior surface of a vertically sectioned segment.



c.

Fig. d.—Anterior surface of a vertical section 4 mm. behind that shown in Fig. c. The lower of the two lesions is the continuation of the large lesion illustrated by Fig. c.

PLATE I.

Illustrating Le Count's Article, page 85.

these repeated attacks persistent weakness of the right upper limb ensued.

"*Examination.*—The weakness of the right side of the face increased greatly after the man entered the hospital, and the palsy was of the cerebral type, i. e., slight in the upper part of the face and almost complete in the lower part. The weakness of the right upper limb also increased considerably, and the right biceps and triceps reflexes were much exaggerated, whereas the corresponding reflexes on the left side were normal. Stereognostic perception and sense of position were lost in the right hand. Tactile sensation also seemed to be diminished in this part, but was normal in the face. The motor power was about normal in the lower limbs, and the patellar reflex was only a little prompter on the right side. Babinski's sign was not distinct on either side. The ocular examination revealed little that was abnormal.

"The case was clearly one of syphilis of the brain with convulsions of the Jacksonian type, only the right side of the body being involved, and the persistent paralysis being confined to the right side of the face and the right upper limb. Other attacks which the patient had made the case an unusual one and worthy of careful study.

"Independently of the typical attacks he had twitching confined to the right side of the face lasting an indefinite period, an hour or more at a time and occurring very frequently. There were periods in which he had none of these spasmodic movements, but the movements were almost constantly present, and resembled a tic movement or fibrillary twitching. They were not excessive in their intensity, and implicated the muscles about the right side of the mouth, about the right eye, the right cheek, the right ear, and the right side of the neck. After persisting for some weeks in the face they ceased in this part, and were observed in the right upper limb. The whole right upper limb was displaced in these clonic movements, but the displacement was not excessive, and the spasm consisted of slight twitchings lasting indefinitely, as did the previous twitchings of the right side of the face. There was no impairment of consciousness. The peculiarity was the almost constant duration of fibrillary or twitching movements in the face at first and later in the right upper limb, movements that were very unlike the severe spasmodic contractions usually seen in an epileptic attack, both in intensity and duration."

CEREBRAL HEMORRHAGE AND HEMIPLEGIA.

Differential Diagnosis. The differential diagnosis between cerebral hemorrhage and cerebral embolism is often difficult, and a case is reported by B. Stow[1] where cerebral hemorrhage following arteriosclerotic granular nephritis was regarded as cerebral embolism, largely because of the presence of a heart lesion. The patient was a widow, 45 years of age, first seen two months after her monoplegia, which took place while she was crossing a street and was associated with complete unconsciousness. Examination showed "a loud, double murmur at the aortic valve," and an enlarged heart. The right arm was paralyzed and the right leg was weak. The urine showed slight traces of albumin. At the postmortem examination an aberrant chorda tendinea was found below the aortic valves, the folds of which latter were normal. There was no evidence of a present or past endocarditis. In the left hemisphere of the brain, opposite the knee of the internal capsule in the globus pallidus of the lenticular nucleus was a recent hemorrhage about one centimeter in diameter. In the putamen was another about three times as large. At the lateral nucleus of the optic thalamus and pressing on the posterior limb of the internal capsule a third recent hemorrhage was found about one-half a centimeter in diameter.

The differential diagnosis between organic and hysterical hemiplegia is described by C. F. Hoover[2] under the caption: "A new sign for the detection of malingering and functional paresis of the lower extremities." The sign is based on the following facts: If a normal individual, lying on a couch, in the dorsal position with legs extended, lifts one foot, the heel of the other foot always digs into the couch. This complemental opposition, as Hoover terms it, is felt by placing the hand under the tendo Achillis. In case of paralysis of one leg, due to a pathologic lesion, if the patient attempts to lift the paralyzed foot, there is the

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(1) Medical Record, May 15, 1909.
(2) Jour. Am. Med. Assoc., Aug. 29, 1908.

same complemental opposition and the heel of the sound side digs into the couch. If the patient lifts the sound foot the complemental opposition of the paralyzed limb will depend on its strength; there will be no movement whatever if that limb is completely paralyzed. Just the opposite takes place in case the paralysis of the leg is hysterical or simulated. If the patient be told to lift the paralyzed leg, or to make a great effort to do so, there will be no movement of the paralyzed limb (provided the paralysis seems to be complete) nor any complemental opposition of the sound one. If, however, the sound limb is lifted, complemental opposition is exhibited in the paralyzed limb and the heel digs into the couch.

In cases of organic hemiplegia or crural monoplegia the phenomenon of complemental opposition on the healthy side never fails according to Hoover. On the other hand in two cases of traumatic crural monoplegia as above noted, the movement of complementary opposition on the sound side did not take place at all, but when the *sound* leg was raised by the patient, the heel of the presumably paralyzed leg could be felt to press downward upon the bed-clothes. In a case of hysterical hemiplegia Hoover also noted the absence of the movement of complementary opposition in the sound leg when the patient was ordered to move the paretic one. The same sign was found also in a case of astasia-abasia.

These facts established fairly surely by Hoover were later confirmed by P. Zenner,[1] who reports two interesting cases. The first case was that of a young man, 20 years of age, who had a tumor of the pons. "The examination for the sign under consideration was made on the patient at a time when the right leg was absolutely paralyzed, while the left one was apparently normal. When he lifted the left, the sound leg, there was not the least movement of the other; but when he was told to try to lift the paralyzed leg, while it did not move in the least, the heel of the other foot dug into the bed."

The second patient, a married woman of 28, presented an hysterical right hemiplegia and hemianesthesia. "When this patient was told to lift the right, the paralyzed leg, or at least to make every effort to do so, there was no

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(1) Jour. Am. Med. Assoc., Oct. 17, 1908.

movement whatever, either of that leg or of the other; but when she lifted the sound leg, the heel of the paralyzed one dug into the bed." Though this latter movement was at times slight, still the biceps, semimembranosus and semitendinosus of the paralyzed leg were felt to contract forcibly.

These facts observed in America are the basis of a paper upon the subject of J. Lhermitte.[1] While he gives to Hoover all due credit and also refers to P. Zenner's article, he calls attention to the work of Grasset, who in 1907[2] reported as a sign of organic paralysis of a lower extremity the possibility of raising the paralyzed limb alone, without the possibility of raising both lower extremities simultaneously. Babinski, too, has studied associated movements extensively, but he never hit upon Hoover's sign.

Lhermitte has examined into the sign of complemental opposition [This seems the best term to apply to the new sign.—ED.] both among normal and pathologic individuals. It was found constant among the normal. Fourteen organic hemiplegias in the Salpêtrière, in the services of Raymond and Déjerine were examined, and when energetic attempts to raise the paralyzed leg were made, the sign of complemental opposition in the sound leg was always present. Indeed the greater the paralysis, the more clearly the sign is evident. Individuals who had largely recovered their power showed the sign less pronouncedly. There was no apparent relation between location of the lesion or its duration, and the intensity of the sign. In a case of hysteria in a young married woman, 21 years of age, with an hysterical hemiplegia and contractures, the sign failed; and Lhermitte affirms that when contractures accompany an hysterical hemiplegia, the sign will be found to follow the law in organic hemiplegia. There being no postmortem in the case cited, even though the description of the case seems to stamp it as hysterical, robs this exception made by Lhermitte of its value.

Quite the reverse is it in cases of flaccid hysterical hemiplegia. Here the findings are absolutely confirmatory of those of Hoover. Lhermitte concludes that "the absence

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(1) La Semaine Méd., Nov. 25, 1908.
(2) Rev. Neur., March 30, 1907.

of the sign of complemental opposition seems to be related to the functional origin of hemiplegia."

The *diagnosis of hemiplegia in coma* is greatly facilitated by noting the absence of the corneal reflex, which is gone on the paralyzed side in the coma of hemiplegia, but is present in coma from other causes.[1] A case in point, leading to a correct diagnosis, is instanced. A patient, thought to be a victim of morphin poisoning, in coma, the body being wholly motionless, showed a complete abolition of the corneal reflex on the left side, and its persistence on the right; hence a diagnosis of hemiplegia was made. The same disappearance of the corneal reflex occurs in Jacksonian epilepsy. The author concludes that unilateral disappearance of the corneal reflex permits the differentiation of hemiplegia from toxic comas. In chloroform narcosis, for example, *both* corneal reflexes are absent.

Prognosis. The duration of life after extensive hemorrhage of the brain is discussed by W. G. Spiller.[2] According to him life does not terminate within five minutes or so of a cerebral hemorrhage. Of 13 cases of extensive brain hemorrhage of which he has the brains in his collection, life had been prolonged in all at least hours after the onset of the apoplexy, even when the hemorrhage was very extensive, and had broken into the ventricles, and filled all of them, even the fourth. In two cases of this latter sort life was prolonged several hours, in one about five hours, and in the other from eighteen to twenty hours. As a rule extensive rupture into the lateral ventricles is followed by more rapid death than when the ventricles escape, but a moderate hemorrhagic exudate into the ventricles is not necessarily rapidly fatal. It seems to be held by some that life must be terminated within a few days if the hemorrhage has destroyed a large part of one cerebral hemisphere, but in one of Spiller's cases a clot, partly encapsulated, 7x2.5 cm., destroying the lenticular nucleus, a large part of the posterior limb of the internal capsule, and the island of Reil, did not produce death for two months.

Treatment. The treatment of hemiplegia *by physical*

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(1) G. Milian, Le Progrès Méd., May 1, 1909.
(2) Jour. Am. Med. Assoc., Dec. 19, 1908.

exercises is advocated by P. Kouindjy[1]. Long ago in this condition rational exercise and methodical massage were recommended as the only therapeutic measures of value extant. During the last five years in the "Charcot Clinic" at the Salpêtrière 34 cases of hemiplegia were treated by motor reëducation. Three per cent. were cured, 17.7% greatly improved, 64.7% considerably improved, and 16.7% doubtful. The treatment is begun by methodical massage, following a methodical exploration of the paralyzed muscles, in order to determine the tonicity of the different groups of muscles, and of their antagonists. This information being gained, massage of the hypotonic muscles is begun with superficial and deep effleurage, followed later by percussion, and petrissage. The hypertonic muscles, which lead to contractures, are left alone.

Active and passive movements are next made use of. These may be very painful but they are valuable. The hemiplegic hates to move his paralyzed arm, and so he does not; but he is determined to walk, and so he walks. This is really the sole reason why return of power in the leg antedates the return in the arm. Passive motion should be begun early to avoid joint inflammations. Active movements are very important, and show us how to proceed to reëducation of the muscles. Reëducation properly followed out, ought never to fatigue the hemiplegic. Its aim is to transform an existing movement into a normal one, and concrete examples are given. Very important is the reeducation of walking. Instead of putting the healthy foot forward first and then of dragging the paralyzed leg after, the paralyzed limb should be first thrown forward. Various devices, such as walking chairs, traction apparatuses, and inclined planes, should be made use of.

The *surgical treatment* of athetosis and spasticities by muscle group isolation is suggested by S. I. Schwab and N. Allison[2] in a preliminary paper, three cases being recorded, one of congenital spastic hemiplegia in a girl of 12 years, one of spastic paraplegia in a girl of 4 years, and one of spastic diplegia (Little's disease) in a girl of 5 years.

The principles upon which the treatment rests follow: "First, athetosis and spastic conditions of organic origin

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(1) La Presse Méd., June 2, 1909.

(2) Jour. Nerv. and Ment. Diseases, August, 1909.

are essentially similar processes, and athetoid movements may be regarded as variants of permanent tonic spasticities. Second, they may be said to be due to several factors, the more salient of which are, a constantly active set of highly irritative impulses, which arise from cortical motor cells. These impinge upon the muscles which are normally in a condition of slight hypertonus. The overaction of the congenitally stronger muscle group as compared with that of the weaker antagonist group causes this temporary state to become permanent, and there results then a characteristic attitude and deformity. Third, the essential mechanism is highly nervous in origin—meaning by that, that the nerve or nerves supplying the muscle or muscle group by which this state is brought about are the anatomical structures which demand primary attention as the focus of surgical attack.

"In consideration of these facts we have been led to devise a method which we shall refer to as *muscle group isolation*. This implies the isolation of the muscle or group of muscles which are at fault in the production of contracture, deformity or athetosis. It is made effective by cutting off from the central nervous system the connection along which the abnormal impulses, active in causing spasticity or athetosis, are transmitted. This is done by a direct attack upon the nerve itself, by isolating it and injecting it with an alcoholic solution. There has resulted in the cases described an immediate paralysis of the physiologically stronger group of muscles without interfering with the free muscular use of the antagonists. At this point physiological exercise planned further to strengthen the antagonist may be used.

"In the selection of a case in which to try this method for the first time, a simple case of athetosis in which the ulnar nerve was regarded as being primarily involved, was chosen, for the reason that the operation would be neither difficult nor dangerous. Inasmuch as the case presented a median nerve complication, it was an easy matter to inject the median nerve at a later time. Our experience in this instance encouraged us to attempt a more complicated operation in a case in which the spasticity was both more general and more intense. Here the condition was bilateral adductor spasticity of the lower extremities in so-called

Little's disease, requiring an isolation and an injection of the obturator nerve, which supplies the adductors of the thigh, above the division into its branches." The operation was done under ether, the nerve being cut down upon, and 60 minims of alcohol being injected into the sheath. Recovery was prompt, and the results obtained excellent.

Special Problems in Cerebral Localization.

Aphasia. A long and exhaustive article on aphasia, showing the domination of the teachings of Pierre Marie, is contributed to the literature of the year by F. Moutier.[1] The term originally introduced by Broca when he was a surgeon, 37 years of age, at Bicêtre, was aphemia. The first patient observed, the famous Leborgne (whose brain is still preserved in the Dupuytren Museum at Paris), could only say "Tan, tan," but his intelligence was in no way impaired. He seemed to lack merely the faculty of articulating words. At the autopsy, though extensive changes were found, Broca regarded the storm center as lying in the left inferior frontal gyrus, and accordingly proclaimed that as the center for speech. Trousseau objected to the term aphemia and made the term of aphasia triumphant. Without going further into the exhaustive description of the historic evolution of aphasia, it may be said that Wernicke demonstrated the existence of two major varieties, sensory aphasia and motor aphasia. Sensory aphasia was next split by Kussmaul into word deafness and word blindness. At the present time, following Déjerine, there are six distinct forms of aphasia:

(1) Motor cortical or true aphasia, in which the patient cannot articulate or articulates badly; presents disturbances in writing, but can copy the printed page in writing; and often shows some trouble in understanding oral or written language. The center involved is the foot of the third inferior frontal gyrus.

(2) Subcortical motor aphasia, in which the white fibers leading from Broca's convolution are cut off, thus isolating it, the patient being unable merely to articulate.

(3) Wernicke's aphasia, in which there is destruction of the first temporal gyrus, the gyrus supramarginalis and

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(1) Gaz. des Hôp., Sept. 12 and 19, 1908.

the gyrus angularis, the patient being merely a chatterer, pronouncing words properly, but substituting them for others (paraphasia), or making up new words (jargonaphasia), and also showing word deafness and word blindness, being furthermore almost incapable of writing or of copying print in writing, though being capable of copying designs and printing letters under printed letters.

(4) Pure word deafness, as used by Charcot, in which there is an isolated lesion of the first left temporal gyrus, the patient being wholly unable to understand spoken words.

(5) Pure word blindness with agraphia, in which there is an isolated lesion of the gyrus angularis, the patient being unable to read written or printed words.

(6) Vocal aphasia, in which there is extensive involvement of the entire Sylvian territory, with both motor and sensory aphasia, the patient being unable to articulate, write, read, or understand language.

Moutier then proceeds to inquire into the evidence upon which the superstructure of elaborated aphasia rests, to some extent echoing what Marie has written.

The inferior frontal gyrus plays no part in the disturbances of speech, he declares as his first proposition. Broca's first patient was Leborgne. This patient showed a pure aphemia without paralysis. At the autopsy there was found extensive softening of all of the Sylvian territory. Broca concluded that the initial center from which he supposed the process to spread, being in the third inferior frontal gyrus, represented the center for speech, a conclusion wholly unwarranted by our present knowledge of pathology. Broca's second patient was Lelong, whose brain is also in the Dupuytren Museum, and this brain today shows no lesion of any kind in the inferior frontal gyrus. All that can be seen is one or two feeble scratches made upon its surface either by a finger-nail or by forceps, in removing the meninges. Thus neither of these basic cases proves anything.

Moutier has found between 1861 and 1906 a total of 304 cases of aphasia in which a lesion of the left hemisphere was found on postmortem examination. Of these 304 cases, 201 cannot be used, 26 because of very imperfect reporting, and 175 because there was more or less involvement of other Sylvian territory than merely the inferior frontal gyrus. Of the remaining 103 cases, there are only 19 favoring the conclusion that Broca's gyrus is the speech center, and 84 cases militating against that conclusion. Indeed, even of the 19 cases, there is not one which is wholly irreproachable. There is, says Moutier, not a single observation in the literature where a lesion, confined wholly to the third left frontal gyrus, has given rise to Broca's aphasia or aphemia.

Of the 84 cases above mentioned, there are 57 cases of aphasia with integrity of Broca's gyrus, and 27 cases of destruction of the center without any effect upon speech. More than this, Burckhardt[1] has unwittingly afforded an experimental proof that Broca's gyrus is not concerned in the production of aphasia. This surgeon, being desirous of producing mutism in idiots with very marked verbigeration, excised the speech centers in two of them. Judge of the surprise of the assistants when, coming out of the anesthetic, the patients spoke as freely and distinctly as before the operation!

Is there any admissible distinction between motor and sensory aphasia? The present idea is about as follows: The motor aphasic is a mute, not articulating at all or else very badly; his vocabulary is very limited, but is correctly used. The sensory aphasic is a chatterer, his words being correctly spoken but incorrectly used and grouped. Both, it is true, may show disturbances of writing, reading and hearing words, but there is a quantitative difference between them, the motor aphasics showing but slight disturbances of this nature, and the sensory aphasics showing marked ones. Closely examined, these concepts are found faulty. Indeed, it is impossible to distinguish at the bedside a case of motor aphasia from one of sensory aphasia, basing one's conclusion on disturbances other than those of articulation alone. In other words, no such quantitative distinction as the one above mentioned can hold.

Any distinction between cortical and subcortical aphasia is at present futile, for where shall the line be drawn? No case exists in which cortex alone is involved, the white fibers beneath being always more or less affected. And furthermore, a lesion of the gyrus angularis does not

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(1) Zeitsch f. Psychiat., XLVII, 1891.

necessarily result in word blindness, unless the neighboring visual subcortical tracts are involved. As for word deafness, Pierre Marie has shown that it does not exist purely, all cases reported being merely ordinary sensory aphasia, with intellectual deficit. Souques has reported a case of destruction of the first left temporal gyrus without word deafness, and Grasset and Rimbaud reported another of the same sort, with softening of the posterior third of the first left temporal gyrus. Burckhardt, already quoted, resected the posterior portion of the third inferior temporal gyrus to cure auditory hallucinations, but he caused not a trace of word deafness. The only thing noted was a paraphasia lasting a week after the operation. Kalischer and also Rothmann, working on dogs trained to perceive a certain tone to gain the reward of meat, found that they lost none of their ability upon unilateral or bilateral resection of the whole temporal lobe, even to the ventricle.

Marie's Doctrine of Aphasia. Moutier, in his 1908 Paris thesis, based on his work with Pierre Marie at Bicêtre, analyzed 23 unreported cases of Broca's aphasia or anarthria, and 3 cases of destruction of Broca's center without aphasia. He goes on to say. "Our material for study comprises, therefore, the different accepted anatomo-clinical types, those cases in which exist motor disturbances alone (pure aphasia and anarthria), and those cases in which motor disturbances are associated with disturbances of writing and hearing of words (Broca's aphasia). Some of these cases were studied both macroscopically and microscopically, some only macroscopically. Among the latter are a case of pure anarthria and six cases of Broca's aphasia with integrity of the third left inferior frontal gyrus; a further group of five cases of Broca's aphasia with softening of the Sylvian territory, including both Broca's gyrus, the lenticular zone and Wernicke's zone; and finally a single mixed case of anarthria due to a lesion of the lenticular zone and pseudobulbar disturbances due to lacunæ in the central gray nuclei.

"By microscopic study of several sections we have been able to demonstrate in three cases the existence of *extensive* destruction of Broca's center in right-handed subjects, without a concomitant aphasia; in one case, absolute integrity of the third inferior frontal gyrus in a case of anarthria;

in three cases, integrity of the foot of the third inferior frontal gyrus in the indisputable presence of Broca's aphasia. A certain number of cases studied in serial sections could not be used for purposes of exact localization, including one case of pure anarthria with simultaneous lesions in both the third frontal gyrus and the lenticular zone, and five cases of Broca's aphasia with simultaneous lesions in the third frontal gyrus, the lenticular zone and Wernicke's zone.

"By way of summary, four times (once in a case of pure anarthria, and three times in clinically indisputable cases of Broca's aphasia), microscopic study showed absolute integrity on the part of the left inferior frontal gyrus from which not a degenerated fiber issued, despite the intensity of the articulatory disturbance. *The third frontal gyrus is therefore to be completely separated from the cerebral language zone.*

"We have tried to account for frequent degeneration of the inferior frontal gyrus, at the suggestion of Pierre Marie, by the arrangement of the branches of the middle cerebral artery. We have found that from the main trunk of the middle cerebral artery the arterioles destined for Broca's convolution often spring singly. They are slender, issue in a direction opposite that of the blood stream in the middle cerebral artery, and run a tortuous course before dividing up into capillaries. It is easy to comprehend how, in these conditions, the least narrowing of the Sylvian artery will favor ischemia in Broca's center before it does in any other region.

"If the inferior frontal gyrus can no longer be maintained as a center with specifically known functions, what conclusions may be drawn from the above statistics? A double conclusion immediately suggests itself: *Each time that there has been demonstrated during the life of the patient pure motor aphasia of the type called subcortical or anarthria, there has been found in the brain after death either softening or hemorrhage in the lenticular zone. Each time that the motor disturbance has not occurred isolated or pure, that is, each time the patient has presented a group of symptoms which characterize Broca's aphasia, there has been found after death a double focus in the brain: one lying in the lenticular zone, the other involving*

Wernicke's zone or the fibers which issue from it. Broca's aphasia is therefore simply Wernicke's aphasia plus pure motor aphasia or anarthria; and moreover, a dstinction between cortical and subcortical motor aphasia cannot longer be maintained.

"Alteration of Wernicke's zone is constant in all autopsies of patients with Broca's aphasia. This is simply a matter of observation. The theory of Pierre Marie easily explains this fact by making of Broca's aphasia a simple Wernicke's aphasia modified by a lesion causing difficulty in the articulation of words; the classical theory cannot explain this fact. Wernicke's territory retains for us its classical limits, vague as they are, in which, at all events, are to be found the gyrus supramarginalis, the gyrus angularis, and the two first temporal gyri.

"Upon a lesion of the *lenticular zone* depends anarthria; that is to say, motor disturbance of articulation. This zone comprises the central gray ganglia (nucleus caudatus and nucleus lenticularis), the internal and external capsules, the island, the fibers from the motor cortex, as well as an important series of association fasciculi of the brain. At the postmortem examinations the spots commonly found involved are those beneath the island of Reil, the lenticular nucleus, the external capsule, the internal capsule, and the foot of the corona radiata. Most usually different systems are simultaneously involved. In front the lenticular zone stops at the foot of the third left frontal gyrus. It has been proved by our serial sections, and by those of Souques, that anarthria may exist without a single degenerated fiber radiating from the lenticular zone towards Broca's center. Posteriorly the lenticular zone terminates at the level of the temporo-parietal isthmus of Pierre Marie. This isthmus is a bridge of white substance, crowded between the bottom of the Sylvian fissure outside and the inferior horn of the third ventricle inside. Any lesion in front of this isthmus causes anarthria; any lesion behind it causes Wernicke's aphasia. It is only necessary for a focus in the lenticular zone to involve even ever so little the territory behind the temporo-parietal isthmus, for the anarthria to cease to be pure, and for Wernicke's aphasia to appear, thus constituting Broca's aphasia."

Clinically, Pierre Marie separates what he calls *intrinsic* from *extrinsic aphasia.* "There exists a single form of aphasia, characterized by internal disturbances of language, that is to say, the aphasia of Wernicke, which is intrinsic, localized in the temporo-parietal territory. In front of this zone cerebral lesions give rise to extrinsic aphasia, which is really not aphasia at all, but anarthria. Behind this zone lesions involving the association pathways from the cuneus will cause *pure alexia. Anarthria,* an extrinsic form, and *aphasia,* an intrinsic form of language disturbance, occur in involvement of the territory supplied by the middle cerebral artery; when they occur together, there is realized Broca's aphasia. *Alexia,* another extrinsic form, is a variety of that aphasia caused by lesions in the back of the brain. It may be associated with more or less intense disturbances of Wernicke's type of aphasia. This form may be called alexia associated with Wernicke's aphasia.

"In sum there are five distinct sorts of aphasic disturbances of language: three are fundamental—Wernicke's aphasia, anarthria, pure alexia; two are syndromes—Broca's aphasia, and alexia and Wernicke's aphasia."

(1) Wernicke's aphasia, in the classically received acceptation of the term and in the classification of Pierre Marie, is identically the same thing. Both its localization and its symptomatology are the same. The Wernicke aphasic is a chatterer, afflicted with logorrhea, paraphasia or jargonaphasia. His talk is, however, fragmentary, punctuated with repeated self-interruptions, interjections, exclamations, affirmative terms. There is no difficulty in articulating words, but often different parts of words are pieced together to make new ones. The Wernicke aphasic always has something to say, whether false or true, correct or incorrect in both form and matter. Paraphrasing is common, nouns being forgotten. Writing is in general profoundly affected, except for the patient's own name, but even this he rarely spells correctly. Attempts at writing often show merely senseless up and down strokes, and the same jargon found in the speech is found in the writing. Neither can printed matter be copied in longhand. The patient *reads* just as he talks, with the same faults; or he may not be able to read at all. He sees a word, and without analyzing its constitutent letters, he

guesses at it and blurts out anything. Often there is great difficulty in the comprehension of words, a veritable word deafness—a term convenient to keep as describing a symptom, but not having the significance as to localization formerly attributed to it. As a rule, the ability to compute little sums is also missing. Lastly, it should be insisted that hemianopsia and hemiachromatopsia should be carefully searched for.

(2) Anarthria is essentially merely a more or less pronounced disturbance in the articulation of words, not dependent upon paralysis of the lips and tongue. Possibly it is a kind of incoördination or ataxia. It often exists pure, in the isolated state, associated with no other changes, in which case it is the anarthria of Pierre Marie. An individual, often young (syphilitic or cardiac) is the victim of a right-sided hemiplegia. When he recovers from his coma or somnolence he cannot speak. His tongue, though not paralyzed, does not obey his will. He understands all but says nothing. There is but one symptom: the suppression of articulate speech. Sometimes monosyllables are preserved, and are used for every attempt at speech. Communication by gesture is perfectly well done.

(3) Broca's aphasia is, according to the Pierre Marie conception, merely anarthria plus Wernicke's aphasia, being therefore a syndrome. It differs from Wernicke's aphasia, which it closely resembles, by the addition of the anarthria. This modifies the paraphasia, jargonaphasia, and the ready speech. The word deafness, alexia and agraphia of the Wernicke aphasic are all present, but usually in lessened amounts in Broca's aphasia than in Wernicke's aphasia. The Broca aphasics are right-sided hemiplegics; the Wernicke aphasics are not paralyzed at all. If the anarthria is intense, there may be mutism, so that word deafness and alexia will be found only on careful examination. If the anarthria is not intense the same use of exclamations, interjections, and fragmented speech seen in pure Wernicke aphasia is evident; but the phrases are not so well executed. At the moment of seizure alexia is usually total in the Broca aphasic, but ability to read is more rapidly gained than in the case of the Wernicke aphasic. Troubles in writing, communicating by gesture, and in computing are all present, but usually in less degree than in the

Wernicke cases. Intellectual deficit is always present in the Broca asphasic, and even a slight examination will show a diminution of the memory, of the powers of observation, of judgment, or orientation. The imagination is weakened. On the other hand, the emotional side is markedly exaggerated.

(4) Alexia depends upon a lesion in the back part of the brain, generally a softening of or hemorrhage into the cuneus, and cutting or destroying the optic pathways on the outer side of the occipital horn of the lateral ventricle. Alexia is rarely absolutely pure, being generally associated at the start with a slight degree of agraphia, paraphasia, etc., just because a lesion causing alexia is almost certain to involve Wernicke's zone. Possibly pure alexia does not exist.

(5) The last form of aphasia according to the Pierre Marie theory is in reality a syndrome, consisting of Wernicke's aphasia plus alexia. As has been said, alexia alone is rare, and most aphasics under class 4 will doubtless be found to belong to class 5 when carefully analyzed. An alexic with Wernicke's aphasia can neither read nor copy, though he may write spontaneously or upon dictation. Yet he cannot read what he has written. He cannot understand what is said to him, and may be paraphasic.

Prognosis in Aphasia, Reëducation. The different forms of aphasia are capable of regression, but in different degrees. (1) Cases of anarthria are susceptible to reëducation, because in these cases intelligence is intact, thus greatly facilitating the cure. At the end of six months to a year the patient may have almost entirely recovered, but it is exceptional for recovery to be absolute. (2) In the Wernicke aphasics reëducation is in general very slow, and always more incomplete than in anarthria. The most fugacious symptom is the word deafness, and it often disappears without leaving any traces of its former presence behind it. The alexia always improves somewhat, even though only a little. The agraphia, on the other hand, tenaciously persists. If the paraphasia is not very extensive it may almost completely disappear.

(3) The prognosis in Broca's aphasia is extremely variable. There are no two cases alike, the relative proportions of anarthria and of Wernicke's aphasia in this syn-

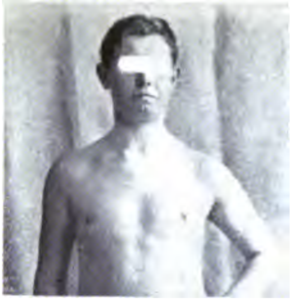

Fig. a.—Hn. after operation. "Voluntary" movement of the left upper limb in placing the hand on the iliac crest.

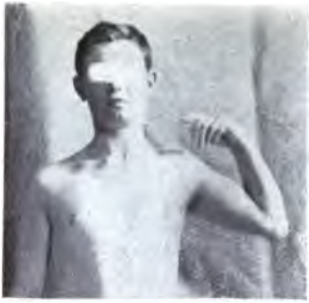

Fig. b.—Hn. after operation. "Voluntary" flexion of elbow and abduction of shoulder. Fingers continuing to slowly flex.

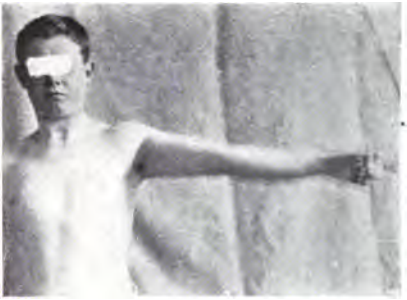

Fig. c.—Hn. after operation. Forcible voluntary abduction and extension of limb, showing the effort causes contracture of the digits. (3 sec. exposure.)

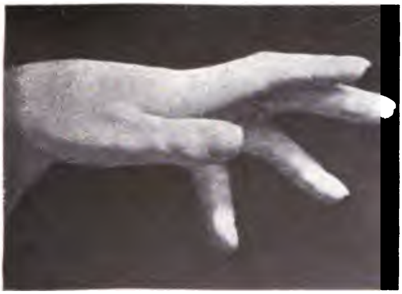

Fig. d.—Hn. after operation. Fullest possible "Voluntary" extension of the digits. (Instantaneous photograph.) Graded power of extension from thumb active, to little finger inactive.

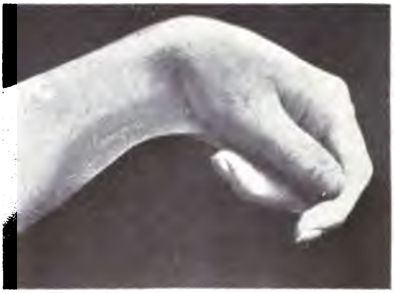

Fig. e.—Hn. after operation. Fullest possible voluntary flexion of digits. the ring and little finger contracted by extra flexion of contracture out of sight. The concurrent hyperflexion of wrist (that is, absent normal extension) is well marked. (Instantaneous photograph.)

PLATE II.

Illustrating Sir Victor Horsley's article on the Motor Area. (Pag
124.)

drome being infinitely variable. Age influences the outlook, which becomes increasingly bad with increasing years. The anarthria has a tendency to retrogress, but Wernicke's aphasia has not. Hence the paraphasia and jargonaphasia tend to persist in Broca's aphasia. It is a question whether recovery can ever be complete. The prognosis in (4) alexia, and (5) the syndrome of alexia plus Wernicke's aphasia is sufficiently evidenced to be passed over.

Diagnosis. The diagnosis of aphasia in general is easy, the principal differential points being pseudobulbar paralysis and senile dementia. (1) Anarthria, when total, may lead to the suspicion of pseudobulbar paralysis. In anarthria the trouble is verbal; in pseudobulbar paralysis it is vocal; and there is paralysis of the tongue in the latter, but not in the former. The anarthritic patient can sometimes say a few words or syllables, and he says them vigorously. The patient with pseudobulbar paralysis can say what he wants to, but he says it with a weak voice and mumblingly. The anarthritic tends toward recovery; the pseudobulbar paralytic grows progressively worse.

(2) In Wernicke's aphasia diagnosis is usually easy except in the old in whose cases senile dementia must be differentiated, and sometimes it cannot be done. Thus in certain old patients with logorrhea, incoherency, dirty actions and habits, difficult to get along with, aphasia at times is hardly more than an hypothesis. If hemianopia can be demonstrated it settles the question, but the intelligence of the patient frequently does not suffice for the test.

(3) The diagnosis of Broca's aphasia is helped by the right-sided hemiplegia, absent in pure Wernicke's aphasia. It is to be made whenever anarthria exists in company with Wernicke's aphasia. The admixture of these two elements is in any proportion, thus giving rise to various groups of cases. In group (a) the anarthria predominates, the patient being able to pronounce only a few isolated words, but he can understand all except long phrases and sentences, can write somewhat, read a little, and he shows but little or no paraphasia. In group (b) the anarthria is dominated by Wernicke's aphasia. In group (c) the admixture is approximately equal. Finally, a last group (d) exists where all the faculties of the patient are involved. He may be mute, or he may be capable of repeating one

word or interjection over and over again. He cannot read, write, or comprehend a single word. This complex is still aphasia, but it is "total aphasia" in the old sense. Moutier designates this form as *aphasia maxima of Broca.*

According to the classical teaching, aphasia is a sensory disturbance; according to the teaching of Pierre Marie it is an intellectual deficit. According to the first theory the patient is admitted to be really deaf and blind to spoken and printed words only; according to Marie's theory the patient is not capable of using what he really hears and sees, but does not comprehend. There is in the aphasic a general intellectual deficit, and a specific intellectual deficit for the comprehension of language in any form. The general intellectual deficit involves descriptive and emotional gestures, memory, association of ideas, judgment and attention. The specific intellectual deficit involves the reading of printed or written words, writing, and the comprehension of spoken words. The disturbances of language appear, unfold, and recede in conformity with the usual laws for intellectual disorders, the most recently acquired being the first to go. Thus nouns disappear before verbs, and the adjective also before the verb; or in a polyglot, the most recently learned language is the first to disappear. There is no *real* deafness in aphasics. Sounds, signals and words are *heard,* but not interpreted. Though they *seem* very intelligent in some instances, even cursory examination shows them to be lacking in intelligence.

The present status of the question of aphasia is also ably reviewed by H. Liepmann.[1] He concludes that the fundamental conceptions of Broca and Wernicke still hold, and that upon them the present doctrine of aphasia can still further build.

Clinical Cases of Aphasia. A case of aphasia which bears out the theory advanced by Pierre Marie is reported by Anglade and Latreille.[2] The patient originally consulted Anglade in March, 1904, because she "could not say what she wanted to." The onset was abrupt in the preceding November, but without a stroke or loss of consciousness. Improvement followed continuously until October, 1906,

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(1) Neurolog. Centralbl., May 1, 1909.
(2) Jour. de Méd. de Bordeaux, Aug. 23, 1908.

when disturbance of speech again took place, together with mental enfeeblement and loss of memory. At this time she talked a great deal, but her language was incomprehensible. The vocabulary was rich, the words clearly pronounced, but without sense. One example suffices: "What is your name?" A. "Mon Dieu! my paper; that's unfortunate; it's his thing; I can't; ah! my paper; if I only could!" A diagnosis of sensory aphasia and senile dementia was then made, with the lesion probably located in the zone of Wernicke, following Marie's scheme.

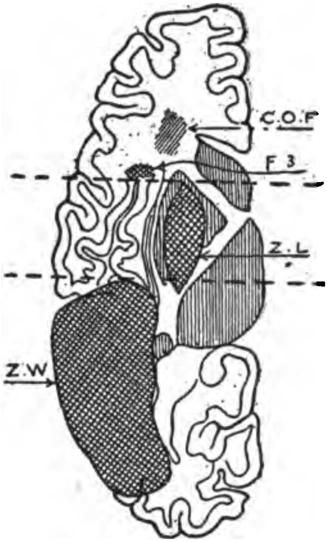

Fig. 5.—Left hemisphere (Flechsig section). C. O. F., frontal portion of centrum ovale somewhat softened. F 3, a very slight patch of yellow softening. Z. L., lenticular zone. Z. W., Wernicke's zone. Between the two dotted lines is situated the quadrilateral area of P. Marie.

In February, 1907, the patient had an apoplectiform attack, remaining comatose one day. When she recovered she was completely paralyzed on the right side, and she could not talk. She could not reply to a single question. She smiled at the sight of food, but all she ever said was "Oh! zeu." This condition suggested, according to the old theory of aphasia, a lesion of Broca's gyrus, but it more probably indicated a lesion of the lenticular nucleus compressing or destroying the internal capsule, and so bringing about the disturbances of language called anarthria by Marie.

The postmortem findings made after the patient's death in March are clearly indicated in the accompanying figure 5. In the left hemisphere there were noted: (a) a focus destroying a large part of the inferior parietal, first temporal and the angular gyri. The lesion appeared to be old. (b) In the lenticular nucleus is a recent hemorrhagic focus. (c) The foot of the third frontal gyrus, and the frontal part of the centrum ovale present in very limited points a slight degree of yellow softening. (d) A little above the calcarine fissure there was a band of yellow softening. In the *right* hemisphere there was an area of softening in the area corresponding to Wernicke's zone. Clinically the case was for three years an example of Wernicke's sensory aphasia. Then, being seized with a stroke of paralysis, and losing her speech, anarthria was added to the already existing Wernicke's aphasia, thus giving rise to Broca's aphasia. The postmortem findings seem to confirm Marie's teachings in regard to aphasia.

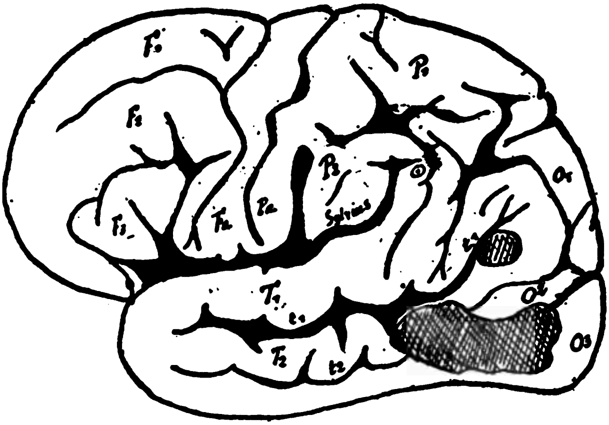

Fig. 6.—Left hemisphere.

A case of aphasia with postmortem findings is reported by R. Dupérié.[1] The patient, a woman of 70 years of age, right-handed, and able to read and write, was the victim of two strokes separated by a slight interval in 1906. From the first stroke her hemiplegia resulted, and speech disturbances and mumbling rapidly followed. After the second stroke the disturbances of speech were marked. For two years the only words the patient could say were "yes" and "no" and the monosyllable "tou," which she repeated without purpose as the sole expression of her desires and answers. She was absolutely incapable of reading print or writing; incapable of writing or of tracing with her

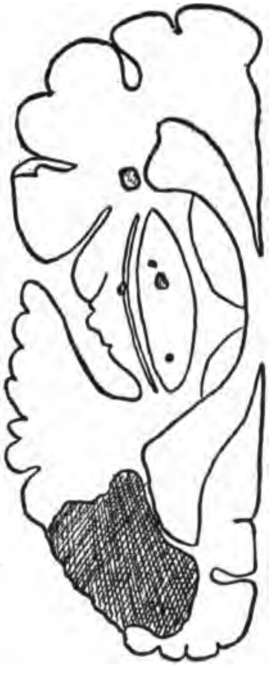

Fig. 7.—Left hemisphere. Section beneath a Flechsig section.

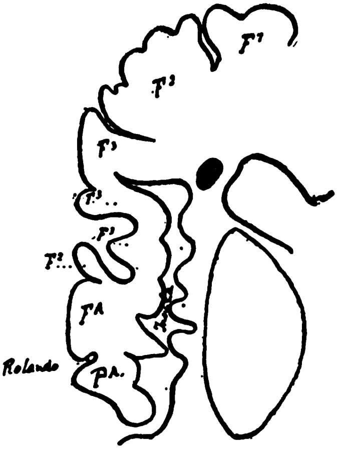

Fig. 8.—Left hemisphere. Section through the frontal lobe.

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[1] Jour. de Méd. de Bordeaux, Feb. 21, 1909.

left hand anything resembling a letter or figure; incapable of telling time, repeating words or of making the most simple gestures. Word deafness was undoubtedly present. She could not sing words, but she hummed monotonously, making use of the monosyllable "tou," and making up her notes as she went along. She recognized those about her, even those whom she had not seen for a long time, and was able to express her emotions by her facial play and tears. Thus she remained until November, 1907, at which time marked mental deterioration set in, and she became exceedingly dirty in her habits. In the following September (1908) she died of pneumonia.

The postmortem findings are interesting. Evidently hers was a case of Broca's aphasia, or anarthria plus Wernicke's aphasia, if Marie's concept be correct. There was found a very intense atheroma of the basal arteries, and of the two middle cerebrals. The left middle cerebral for a centimeter and a half was completely impermeable. The basilar artery was very much calcified. The left hemisphere (see figure 6) showed two foci: one in the gyrus angularis, and the other in the second temporal and the second and third occipital gyri. A Flechsig section showed these same foci. A cut just beneath the Flechsig is shown in figure 7. Noteworthy are the lacunæ of disintegration in the lenticular zone. A section made at a still lower level is shown in figure 8, which sufficiently explains itself. Thus the portions involved are the lenticular zone of Marie and Wernicke's zone, which give Broca's aphasia. Very noteworthy is the fact that the third inferior frontal gyrus was found macropscopically wholly unaffected.

SENSORY DISTURBANCES OF RADICULAR TYPE.

The symptomatic value of sensory disturbances of radicular type in lesions of the cerebral hemispheres forms the subject-matter of a long and thorough paper by Lhermitte.(1) Up to a short time ago it appeared to be impossible to come to any conclusions as to the site of a cerebral lesion based only upon sensory disturbances in the periphery of the body, except in cases of hemianesthesia. But even in the latter case a topical diagnosis is precarious. More recently, however, a series of observations tends to show that in certain cases at least anesthesias caused by purely cortical lesions exist, and that these anesthesias closely resemble the topographical distribution found in disturbances of the spinal cord, in that they are radicular in type.

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(1) La Semaine Méd., June 16, 1909.

The first of these cases of this sort of anesthesia in cortical lesions was observed by an American, F. S. Madden,[1] whose article is referred to. In this case there was present an anesthesia of the ulnar type, or of the radicular type at the level of the 8th cervical and 1st dorsal segments of the cord. The postmortem examination showed a cystic tumor compressing the right first central and superior parietal convolutions. This is a case showing, as it were, merely the rough draft of a radicular type of cerebral anesthesia. A second case was reported by Klein,[2] in which, following a blow on the head, among other symptoms there developed a hypesthesia involving the radial side of the fore arm, such as occurs in a lesion involving the fifth and sixth cervical segments of the cord. Two more cases have been reported by Bonhöfler in the same volume of the *Deutsche Zeitschrift für Nervenheilkunde* as the one containing Klein's report.

Disturbances of sensibility in the course of cerebral disseases, then, may present a topography essentially radicular in type, and may affect not merely the hand, but the forearm as well. This conclusion is not new, being already drawn by C. K. Mills and T. H. Weisenburg,[3] to whose paper Lhermitte refers. In the case of these latter observers a patient 38 years of age, struck over the right parietal region, showed two weeks later an inability to use his left hand correctly, doubtless because of hypesthesia to all kinds of sensation in the forearm and hand. This hypesthesia gradually disappeared, leaving two years later a band stretching from the elbow along the forearm, and involving the last two fingers, giving the radicular distribution of anesthesia seen in a cord lesion at the level of the 8th cervical and 1st dorsal segments. Even the lower extremities may show such segmental distribution of anesthesia, as is demonstrated in a case reported by Goldstein.[4]

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(1) Jour. Nerv. and Ment. Diseases, February, 1893.
(2) Deut. Zeit. f. Nervenheil., 1904, Vol. 26, p. 327.
(3) Jour. Nerv. and Ment. Diseases, October, 1906.
(4) Neur. Centralbl., February, 1909.

The anesthesia involved the territory supplied by the sacral segments and the last lumbar segment of the cord.

It has been suggested that such distribution of sensory disturbances is due to hysteria, but the localization of the anesthesia is wholly different from that of hysteria. Goldstein asks whether it is not possible to consider such radicular anesthesia as the exaggerated expression of a normal phenomenon, viz.: the unequal excitability of cutaneous radicular zones. Supposing a cerebral lesion to lower the threshold value relatively of one of two symmetrical areas of skin, it might, says Goldstein, be conceivable that one area would be felt to be normal and the corresponding area hypesthetic. Lhermitte believes the cerebral lesion itself to be the cause of sensory disturbance, and among others he mentions Mills and Weisenburg as sharing this opinion. All the facts recorded suggest that the cerebral localization of these sensory centers is in the cortex or the layers immediately subjacent. The exact cortical centers of the different cutaneous areas Lhermitte does not attempt to locate.

MOTOR AREA.

The function of the so-called motor area of the brain formed the subject of Sir Victor Horsley's recent Linacre lecture.[1] After a preliminary account of the history of development of our knowledge concerning the motor area, special attention being devoted to the subject of unipolar excitation during surgical operations, the myelinization method of Flechsig and the anatomical investigations of Campbell, he reports the first case of absolutely pure lesion of the gyrus precentralis which he ever met with. The case was that of a well developed boy, Hn., of 14 years, who at the age of 7 began gradually to show athetoid movements of the left hand, and later on violent convulsive movements of the whole upper limb, in which the arm was usually strongly flexed and adducted in jerks across the trunk, and more rarely flung out in abduction. The elbow was fixed in semi-extension, the forearm strongly pronated, the wrist flexed, and the fingers either in an interosseal position or flexing and extending independently. The movement was worse on walking, and when his attention or the attention of others was drawn to it. When the limb was quiet his purposive or voluntary movements were normal and powerful. The reflexes, superficial and deep, were everywhere normal; his sensation—all forms—was also normal.

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[1] British Med. Jour., July 17, 1909.

Operation was undertaken for the purpose of stopping the athetoid and clonic spasms by excision of the corresponding motor area involved. The cortex was laid bare, and then stimulated with a Kronecker graduated coil furnished with three dry Obach cells. The gyrus precentralis was then thoroughly explored, all the movements obtained involving those of the arm. "No other part of the cortex in the neighborhood giving any response in the upper limb, the gyrus thus marked out was excised very carefully by making a vertical incision through the pia mater along the middle of the surface of the convolution, reflecting the pia mater to the sulci on each side, and gently separating it to the bottom of the sulci so as to permit of excising the whole depth of the gyrus precentralis without any injury to the neighboring gyri or even to the vessels in the sulci, beyond, of course, the laceration of the smallest branches entering the portion of gyrus removed." That this gyrus was the site of the arm centers was shown by the gratifying fact that "the spasmodic movements totally disappeared from the moment that the gyrus was removed, and have remained absent not merely during the short period of total paralysis of the arm, but to the present time—that is, during thirteen months—and that since a month after the operation purposive (that is, voluntary) movements have returned and are developing in efficiency, in spite of the fact that the Betz cell area for the upper limb has been removed.

"Briefly summed up, the effects in Hn.'s case of the removal of the so-called motor centers for the upper limb in the gyrus precentralis may be stated as follows:

"*Immediate.* Disappearance of spasmodic movements.
Complete loss of voluntary movement of the left upper limb.
Postaxial and proximal atopognosis of the same.
Complete astereognosis of the left hand.
Moderate anesthesia to all forms of sensation of the left upper limb, maximal at the periphery.

Remote (that is, a year later). Permanent absence of spasmodic movements.
Partial recovery of voluntary movement of left upper limb.
Proximal atopognosis of left postaxial fingers.
Astereognosis of the left hand.
Slight tactile anesthesia of the ulnar periphery of the left hand."

Changes in sensation observed in Hn. after excision of the gyrus precentralis were elaborately studied by Horsley. "The temporary character of the severe effects observed directly after the interference proves that the sensory representation of the upper limb extends over a wide area of cortex—namely, at least both Rolandic gyri. Practically these provisional phenomena disappeared within three to four weeks after the operation, and, therefore, as fifteen months have now elapsed, it is possible to determine accurately what permanent change or loss of function is coincident with the removal of the gyrus precentralis.

"Position of the limb. During the first twelve days after the operation the limb was motionless and flaccid. The patient was imperfectly aware of the position of the limb, and felt a 'funny numbness' all down the left side, and mostly in the leg. At the end of the first fortnight he knew the general position of the whole limb by reason of his appreciation of the contrast between the temperature and roughness of the blanket and the surrounding objects. Nine months later, if the limb were kept at rest for some time, he lost knowledge of its position. He now (June, 1909) knows the general position of the limb in the dark." The position of the digits was correctly described, but not correctly imitated by the right hand until the third week. During the first fortnight atopognosis was very marked, but as the recovery of movement appeared, the patient's atopognosis disappeared. Fifteen months after the operation the sign was still present. Slight loss of tactile sensibility has been present throughout. Fourteen days after the operation a test tube of water heated near the temperature of the hand was not felt on the terminal phalanx, was occasionally detected on the middle phalanx, and well appreciated as warm on the forearm. The cold tube was recognized as cold everywhere.

The most profound sensory change produced by the removal of the gyrus precentralis was inability to recognize the form of objects—astereognosis. In view of the current hypothesis that the postero-parietal lobule, or the gyrus postcentralis, is the center for stereognostic perception (Cf. Mills, Bruns, Redlich), it was especially important to note what alteration occurred in the case of Hn. He could recognize nothing (nail brush, prayer book, bottles, coins, knives, pipe, matchbox) when the objects were placed in his hand, and even when the fingers were pressed over them, though he once guessed a tumbler to be a bottle, because it was cold. At the present time the astereognosis is extremely marked. In conclusion it may be said that "It is quite clear that the so-called motor cortex is a sensori-motor structure, of which the motor element is the principal funnel-like outlet for afferent impressions coming from many parts, especially the gyrus postcentralis and optic thalamus."

As to the gyrus precentralis, "In view of Campbell's and Brodmann's demonstrations of its richness in granules and its architecture generally, together with the fact that I have just stated, that lesions of both central gyri produce far more sensory disturbance than lesions of the precentral gyrus alone, I think we may safely conclude that the postcentral gyrus is part of the cortical area in which the sensory representation of the upper limb is located. Inasmuch as the sensory properties of the upper limb are all directed to the efficient action of its muscles, it is also not assuming too much to say that, like the precentral, it is a center for tactility, topognosis, muscular sense, etc. As regards Mills' claim, that it is the sole center for stereognosis, this clearly cannot be justified, as my present case and others in which the lesions did not involve the postcentral gyrus prove.

"Just as the disturbances of sensation which follow destruction of the Rolandic gyri are clearly due to loss of the precentral as well as the postcentral convolution, so it is now also certain from the case of Hn. that the precentral gyrus is not in man the only outgoing or motor center for the voluntary movements of the upper limb."

Changes in motion. (a) Consensual movement. "The arm immediately after the operation was perfectly motionless, but by the fourteenth day, when on my request the right (normal) hand was strongly clenched by Hn., his *left* forearm slowly supinated, the wrist slightly flexed, and the fingers also very slightly flexed. (b) Purposive or voluntary movement. At the end of the third week the power to abduct and adduct the shoulder returned. Next, flexion of the elbow, then extension of the elbow, and flexion of the wrist were regained. See figures a, b, c, Plate II.

"Recovery of power in the digits was especially interesting and important. Several days before the movement of the fingers and thumb returned he stated that he felt the power was returning, and that he would shortly move them. The control and exercise of the digits has now attained its optimum in the thumb, which can be slowly flexed and extended, while the fingers as regards extension follow in rapidly diminishing degree; thus, the index finger fairly extends, the middle finger much less, the ring finger hardly at all, and the little finger not at all. As regards flexion, the complication of hypertonia comes in, because the ordinary hypertonia of contracture affects as usual the most paralyzed part, and therefore when the digits are flexed the small fingers curl up into the palm, and the wrist, instead of remaining extended, follows suit. The range of movements are shown in the accompanying photographs (figures d, and e, on Plate II), taken on April 24, 1909. The hand assumes an athetotic posture, and the powerful effort made to keep the arm straight out from the shoulder and to extend the digits is well shown by the movement of the latter throwing them out of focus (3 secs. exp.), as well as by the synergic contraction of the face. Flexion of the elbow is easily performed, though slightly flail-like, and the rotation and pronation movement so well that, as the photograph shows, Hn. can place his hand on the back of the iliac crest.

"Hn. proves for man what has already been established by experiments on monkeys—namely, that so-called volitional movements are not alone generated from the brain through the 'motor' area or precentral gyrus, but must also be subserved by other parts. Further, from what has been already said it is reasonable to assume that, in the absence of the precentral gyrus portion of the arm center, the function of movement is executed by the representation of the upper limb in the gyrus postcentralis from which the fibers descend to the optic thalamus through the internal capsule."

The author's conclusions follow: "It follows from the case of Hn. that:

"1. The so-called motor area of the human cortex cerebri is really sensorimotor.

"2. The gyrus precentralis is in man the seat of representation of (1) slight tactility, (2) topognosis, (3) muscular sense, (4) arthritic sense, (5) stereognosis, (6) pain, (7) movement.

"3. The gyrus postcentralis is in man part of the arm area in which the sensorimotor representation is of the same kind as that in the gyrus precentralis, but in it probably provision for sensorial coördination is greater, and that for efferent impulses less.

"4. The giant pyramids or Betz cells are not essential for the performance of purposive or voluntary movements.

"5. Purposive or voluntary movements can be performed after complete removal of the corresponding part of the gyrus precentralis."

CEREBRAL TUMOR.

Diagnosis by Brain-Puncture. Brain-puncture of late is being advocated for the diagnosis of brain tumor by some, and discouraged by others. E. Forster[1] reports a case of cerebral tumor where this method was not used because of the refusal of friends to permit it, where it might have been used to great advantage. Krause[2] makes the following objections to its use: (1) It is not always easy to find the puncture hole with the needle, so that it may be necessary to cut down on the bone, raise the periosteum, and find it. (2) There is danger of hemorrhage, especially from the large dural veins, and from the meningeal artery, one case of the latter accident coming in Krause's experience. (3) There is special danger where an angioma is present. (4) There is danger of infection in brain abscess, and also in tuberculosis. (5) Finally, the symptoms may be much aggravated by the procedure. Oppenheim,[1] though an advocate of brain-puncture, reports a series of cases where it was of but little assistance: (1) A case where trephining and puncture over the right occipital lobe and cerebellar hemisphere showed no tumor fragments in the brain cylinder, where after death a tumor was found in the right cerebellar hemisphere; (2) another case where brain-puncture was done without avail in several places, cerebellar symptoms then arising leading to operation without finding any tumor, and its discovery after death upon the tentorium between the occipital lobes; (3) a final case, where brain-puncture was done over the left cerebellar hemisphere without result, an operation over the left temporal lobe without result, and at the postmortem a tumor being found in the left frontal lobe.

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(1) Berliner klin. Woch., Feb. 15, 1909.
(2) Berliner klin. Woch., 1908, p. 1,351.

Forster's case was in a woman who 12 years previously had dizziness and headache, with a sort of seizure in which she stared fixedly and discharged the contents of the bladder and rectum unconsciously. This attack lasted two minutes. After it she came to herself and asked: "What is the matter with me?" She has since had two other similar vertiginous attacks. Until Easter, 1907, she was well, when she suddenly lost consciousness and fell against the wall, bit her tongue, showed bloody foam in the mouth, but did not urinate. Later on she became incapable of working because she did not remember what she was doing. Examination of the eyes showed beginning choked disc, the right being more pronounced than the left. The left facial innervation was faulty, and she was somewhat deaf in the left ear. Lumbar puncture showed a pressure of 34 cm., and nothing more of a positive nature. A course of antiluetic treatment gave a negative result. Later a left Babinski toe-sign developed. Her condition gradually grew worse from June, 1908, when she was first seen, until August 20, when she died.

The postmortem showed a tumor of the right frontal lobe, with a pronounced hydrocephalus, and marked flattening of the gyri. From the anamnesis and physical findings one would expect something limiting the intracranial capacity, whether tumor, hydrocephalus, or cerebral syphilis.

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(1) Berliner klin. Woch., 1908, p. 1,301.

Hydrocephalus could in no way be ruled out of the differential diagnosis. Though a tumor might be present, it was impossible to say that it was, or to localize it. Indeed, it would be hard to say whether it were left-sided or right-sided, but later the Babinski reflex suggested it as being on the right side, as did also the negative sign of absent speech disturbance. The author feels that a diagnosis of a tumor in a silent area would have been the best diagnosis, in which event brain-puncture would infallibly have been done over the right frontal lobe, and the tumor doubtless have been located. The tumor is illustrated in the accompanying figure 9. It could easily have been removed had it been localized.

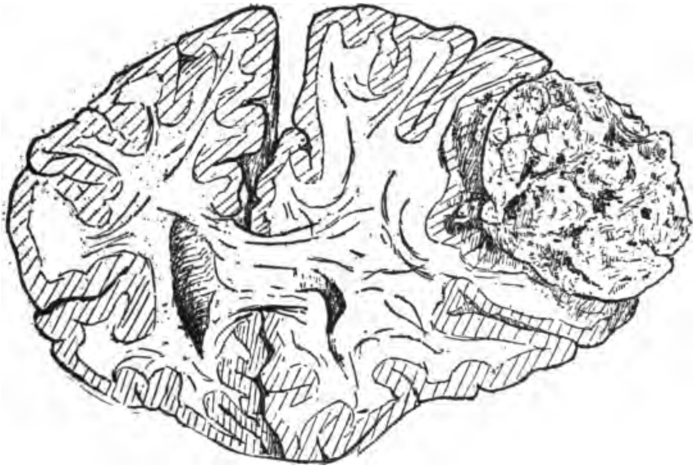

Fig. 9.—Section through the frontal lobe just cutting the most anterior portion of the ventricle. $^{65}/_{100}$ natural size.

The author concludes as follows:

"(1) Brain-puncture should be done in all cases in which the localizing diagnosis of a tumor is uncertain; in all cases where it is necessary to determine the operability of tumors; and in all cases where the differential diagnosis of tumor comes into question in which death is to be expected if nothing is done.

"(2) Brain-puncture ought to be done only after a very careful localizing diagnosis has been attempted, and in the event that no point of departure exists for a localizing diagnosis the silent areas of the brain, beginning with the right frontal lobes, should be examined.

"(3) An operation should not be performed over localities where brain-puncture has shown the brain to be normal.

"(4) Brain-puncture should not be attempted in the case of nontubercular abscesses (with the exception of those cases where possible abscess has not been localized, in which cases the risk of infection must be taken).

"(5) Brain-puncture should be done by neurologists themselves, after suitable arrangements have been made with a surgeon ready to trephine immediately in case need should arise."

Symptomatology. *Optic neuritis.* Concerning the cause of optic neuritis Sir William R. Gowers[1] remarks: "It is maintained by Sir Victor Horsley that increased intracranial pressure is the chief mechanism in the production of such neuritis. He found, and has abundantly proved by experience, that when the pressure is relieved by trephining, whether or not a tumor is removed, the neuritis begins to subside—sometimes quickly, sometimes slowly, sometimes very slowly. The fact is of great practical importance, because it justifies the operation for the relief of pressure even if the symptoms make it unlikely that the removal of the tumor is possible. I do not think it justifies the inference that the intracranial pressure is the cause of the neuritis. We find neuritis in cases in which it is difficult to conceive that the pressure has been increased, and we find it absent in cases in which the increase must have been considerable. But I think we cannot doubt that it is the chief factor in intensifying neuritis and raising it to a degree which inevitably, if left alone, will lead to loss of sight."

Charles H. Frazier,[2] in an address on the surgical aspects of cerebral decompression, with special consideration of the etiology and certain manifestations of papilledema, says: "The conditions in which it has been associated are well

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(1) The Lancet, July 10, 1909.
(2) Jour. Am. Med. Assoc., Sept. 11, 1909.

known, notably tumor, hydrocephalus, and more recently traumatic lesions. It has been assumed by some that the most important factor in its pathogenesis is increased intracranial tension. Plausible as this theory is, it is not borne out by experimental evidence, at least with one or possibly two exceptions experimenters have thus far failed to produce a papilledema solely by increasing intracranial tension."

Frazier made numerous experiments upon animals, from which experiments he derives the following conclusions:

"1. Increase of intracranial tension by artificial means up to a point attended with profound disturbance of the circulatory and respiratory functions was in no instance attended with development of a papilledema, except in two instances not here recorded, and in these there was no marked swelling of the disc, but only a slight edema and filling in of the center of the discs.

"2. The duration of the observation varied from two to ten hours; in one instance increased intracranial tension was maintained and observations made from six o'clock in the evening to four o'clock the next morning. So that the element of time did not affect the results.

"3. In some instances the eyegrounds were bleached (the stage of anemia) and in many there was more or less fulness and occasionally pulsation of the retinal veins, but nothing else.

"4. The degree of intracranial tension artificially produced in these experiments exceeded by far that which obtains in the vast majority of these lesions of which papilledema is a clinical manifestation.

"5. Therefore, in the etiology of papilledema some factor or factors other than increased intracranial tension play an important part.

"6. What these may be is still a matter of conjecture."

Unilateral Optic Neuritis. A case of unilateral optic neuritis resulting from an intracranial tumor is reported by Sir William R. Gowers.[1] The patient, a single woman 37 years of age, began to suffer with headaches when she was 35. Later she found that she was more easily fatigued

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(1) The Lancet, July 10, 1909.

as her headache increased. One day, after a long walk, she fainted and remained unconscious for 2 hours, but without any convulsions; then she vomited for 12 hours. All her life she was liable to faint from any trifling cause. The pain was in the vertex and parietal region, and often in the occiput. In June, 1908, the sight of the left eye began to fail, and in July the left arm and leg grew weak. Her mental state was clear and normal. On examination optic neuritis was found in the right eye only. The left disc was not inflamed, and its appearance showed that it never had been. Smell was normal on the right side but absent on the left. Movement of the eyes was normal, and there was no nystagmus.

"The left wrist-jerk was greater than the right, and so was the knee-jerk, but there was no foot-clonus. The plantar reflexes were both flexor, but the left was less easily elicited than the right. This was true also of the abdominal reflex. Thus the superficial reflexes were diminished, the 'deep' reflexes were increased. That is the general rule when there is some interference with the pyramidal tracts on one side, causing a slight degree of degeneration. But it is noteworthy that the plantar reflex was only altered in degree. The pupils acted to light on each side, but on the left the action was not sustained; the contraction was immediately followed by dilatation. The right optic disc was completely hidden beneath a prominent swelling which occupied its position, finely striated, redder in the center than on the sides, down which the vessels sloped. A few lines of very fine dots were seen in the retina beyond the swelling, coursing towards the macula. One or two very minute hemorrhages were once seen, but they soon disappeared and were not renewed. The veins were not greatly distended. It was apparently a neuritis of moderate intensity with little tendency to pass into the stage of 'choked disc'; it was a form common in intracranial tumors of slow rate of growth.

"A striking contrast to the aspect of the right eye was presented by the left. There was no trace of neuritis present or past, the disc and cup were sharp and clear, the larger vessels of nearly normal size, and the disc presented a grayish rather than a rosy tint, as if atrophy were commencing. A small central scotoma existed for both white and colors, absolute up to 10°. Very dim vision existed round it up to about 30°. With this eye the patient could make out none of Jäger's test-types. Thus the condition was that with which we are familiar in retro-ocular neuritis. The patient's state remaining the same, operation was performed by Sir Victor Horsley on October 13, but two hours later she died. The postmortem showed a tumor between the frontal lobes and the orbital plates, 2⅝ inches from side to side, 2½ from before back, and 1⅝ in thickness. Its structure was that of an indurated endothelioma.

"The chief diagnostic lesson the case has for us is the opposite affection of the two eyes and the manner in which it was brought about. In any case which presents this the condition will probably warrant the same diagnosis. You may assume that the optic neuritis produced by an ordinary intracranial tumor is always produced in both eyes. One may suffer a little sooner than the other, but as soon as it is unmistakable in the eye first affected signs of it can be detected in the other eye.

"The compression of the left optic nerve by the growth was much greater than that of the right. It occurred at the very short piece of the nerve in front of the chiasma, before the nerve passes through the optic foramen on its entrance into the orbit. This was, indeed, longer than usual in consequence of the displacement of the chiasma backwards by the growth. To this greater and earlier constriction of the left nerve must be ascribed the absence of optic neuritis in the left eye and the fact that the symptoms in it were precisely those of retro-ocular neuritis, a central scotoma in the field of vision, and slight signs of atrophy of the disc."

Inversion of the Color Fields. James Bordley and Harvey Cushing call attention to a new and early sign of intracranial tumor, the inversion and interlacing of the color fields. In a series of some 200 cases of brain tumor in which careful perimetric examinations have been made of the visual fields it has been found that color inversion, or interlacing of the color fields—a sign thought to be characteristic of hysteria—is very common, and, indeed, a usual phenomenon.(1) It is believed that the sign is of value in the early diagnosis of brain tumor. Since it is one of the first symptoms to disappear after a decompression, it is re-

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(1) Jour. of Nerv. and Ment. Dis., September, 1909.

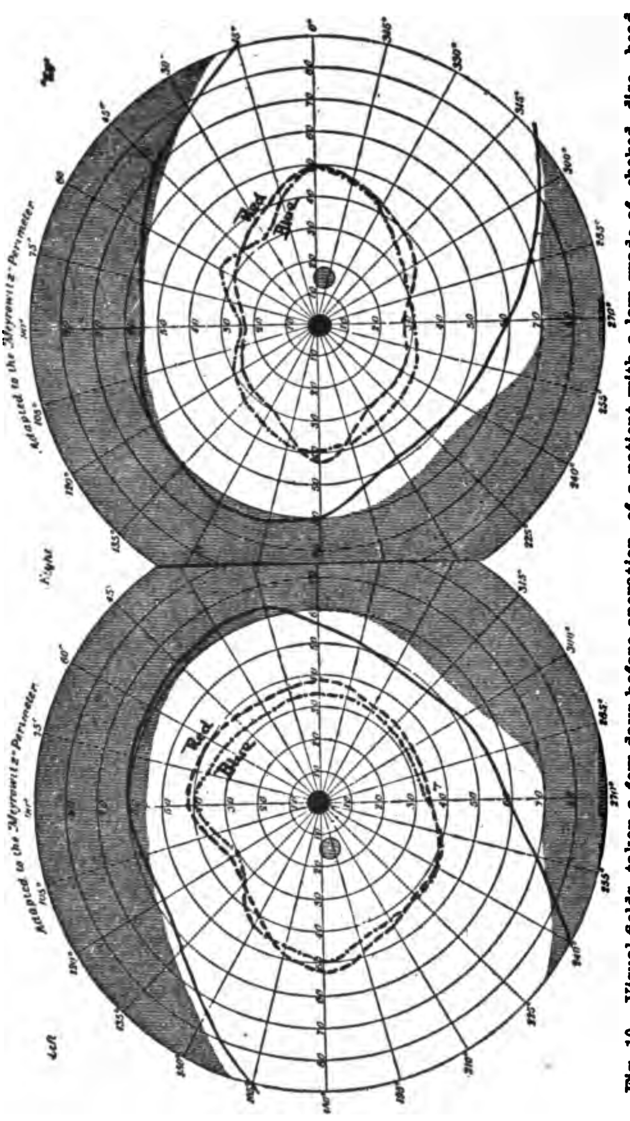

Fig. 10.—Visual fields, taken a few days before operation, of a patient with a low grade of choked disc, headache and focal epilepsy. Note the typical interlacing with considerable inversion of the boundaries for blue, with no alteration of the fields for form (white).

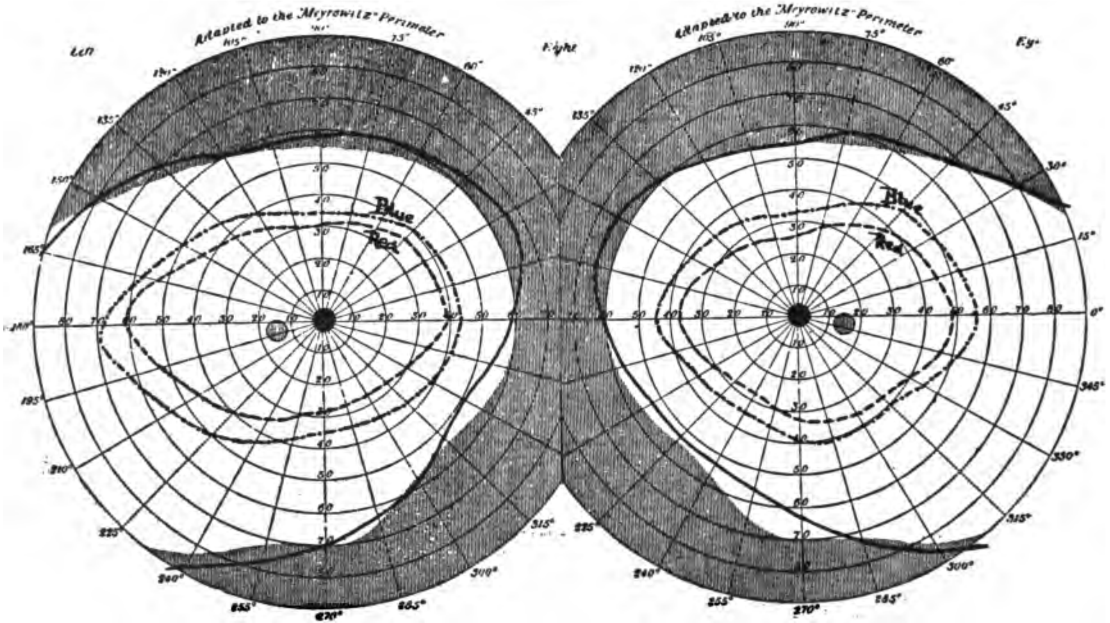

Fig. 11.—Visual fields of same patient eight days after an exploratory craniotomy combined with subtemporal decompression as the tumor was not disclosed. Note restoration of normal color outlines following relief of pressure symptoms.

garded as possibly a prodromal symptom of a choked disc. The accompanying figures[1] 10 and 11 excellently illustrate the symptom and its rapid disappearance after decompression. Cushing, in presenting his paper upon the subject before the annual meeting of the American Neurological Association, held in New York City May 27-29, 1909, said that he wished to give credit where credit was due, for it was Dr. Bordley who first observed and appreciated the possible diagnostic importance of color inversion or interlacing of the color fields in brain tumor.

Pathological Physiology. The pathological physiology of brain tumor in some of its aspects is discussed by H. Cushing.[1] Lack of space forbids more than the author's conclusions, which follow:

"Contrary to the common belief, brain tumors are of frequent occurrence, and possibly there is no disease in which the symptoms are more often overlooked or incorrectly interpreted. For the sake of successful palliative or curative measures, a precocious diagnosis is necessary, and a careful clinical study of all suspected cases at an early stage, with a more intimate knowledge of the local and general pressure phenomena, will tend toward this desired goal.

"A more widespread familiarity with the early appearance of the neuroretinal edema and congestion is needed, and also the understanding that this condition is merely a stage in the process of choked disc. Inversion or interlacing of the boundaries of the color fields, heretofore regarded as pathognomonic of hysteria, has been found to be a fairly constant early phenomenon in tumors.

"One recognized characteristic of the brain under pressure is its tendency to herniate through a cranial defect, and as there is normally an opening at the foramen magnum, a certain degree of protrusion is usually present there. In the presence of such a condition, the withdrawal of the cerebrospinal fluid from the spinal meninges by a lumbar puncture is often hazardous, as it may tend to a sudden wedging of the bulb in the opening, with anemia and paralysis of the vital centers.

"The conducting paths in the brain are more often af-

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(1) Boston Med. and Surg. Jour., July 15, 1909.

fected by simple pressure from tumors than by actual destruction, and the removal of the growth or simple relief from pressure may often lead to a surprisingly rapid restoration of function.

"For an accurate focal diagnosis a thorough, early examination is often necessary, for with an advancing lesion symptoms at a distance may greatly confuse the clinical picture. A considerable percentage of cases that present themselves for surgical treatment are so nearly blind that a study of the visual fields, which should be one of the most helpful localizing signs, is precluded.

"From a histological point of view the classification of brain tumors is most unsatisfactory, and the general behavior and manner of growth of many of them does not conform with accepted views. Some gliomata may be definitely encapsulated and favorable for operative removal, and many tumors of this supposedly malignant type may undergo cystic degeneration or gelatinous transformation."

Clinical Cases. *Temporal Lobe.* The cerebral centers for taste and smell and the uncinate group of fits forms the title of a paper by C. K. Mills,[1] based upon the study of a case of tumor of the temporal lobe with necropsy. The patient was a married woman, 34 years of age at the time of the first examination. The onset was with frontal headache. When she was 29 years of age she began to have slight seizures of a peculiar character, lasting about half a minute. In these spells she was said to have nausea, to be somewhat confused, and to articulate indistinctly and incoherently. She became unconscious for the first time in one of these seizures in October, 1903. In these seizures her face would turn of an ashen hue and her expression become drawn.

"The patient, in describing her attacks in the spring of 1904, said that she had a sensation of some kind in the region of the stomach. This was soon followed by a sensation of taste and then of smell. These were of a definite character in that they recurred in the same way. She could not tell what the taste or smell was, although she sometimes thought that she was just about to be able to do this. These sensations were in some instances accom-

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(1) Jour. Am. Med. Assoc., Sept. 12, 1908.

panied by smacking of the lips, or, as her husband expressed it, clapping of the lips and champing or chewing movements. Immediately after these gustatory and olfactory auræ she became more or less dazed, this confusion being worse at times; in some instances she would keep right on talking through the spell.

"Examination for taste showed that on the back of the tongue she was able to taste correctly. In the chorda tympani area of the tongue she tasted something, but she could not distinguish the quality or flavor of what she tasted. Smell was retained. Examination of her nostrils for motor power, by having her sniff with them, with or without the examiner's finger on them, showed undoubtedly that more force was exerted by the right nostril than by the left. She spontaneously spoke of this difference. The left nostril seemed smaller and thinner than the right. The tongue was protruded in the median line, was not atrophied, and had no fibrillary tremor. The left half of the tongue seemed to be a little smaller and flatter than the right. The left pupil was slightly larger than the right; both pupils responded to light and in accommodation. No nystagmus was present and no external ocular palsies."

In May, 1904, careful examination was made for sensation, motility and reflexes in all parts of the body, the results being almost entirely negative. No ataxia of station or gait was present and no paralysis and no paresis or ataxia of the extremities. Sensation was everywhere preserved. The reflexes also were preserved; the knee-jerks, however, were depressed, more markedly on the left. The bowels and bladder were unaffected. The general condition of the patient was good. Her mentality was fully retained and no aphasia was present. The patient was perhaps at times a little apathetic, but on the whole was in good spirits. She seemed at times in a state of dreaminess.

"My last examination, which was made in August, 1907, never showed any paralysis, disorder of sensation nor hemianopsia. Her husband stated that she was never paralyzed in her face, arms, leg or in any part of her body. Inquiring with regard to hemianopsia, he said that she never appeared to be blind or partially so, either to the right or to the left. She did not run up against things to either side. The only visual phenomenon which he ob-

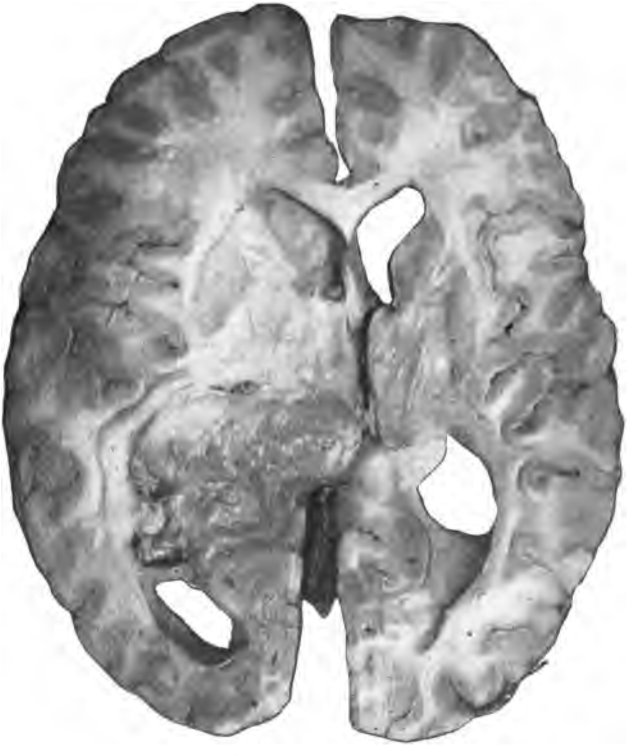

PLATE III.—Photograph of a section at the upper part of the basal ganglia, showing the tumor invading the left thalamus, filling the front part of the posterior horn of the lateral ventricle and implicating the extreme anterior part of the median aspect of the occipital lobe.—C. K. Mills. (Page 138.)

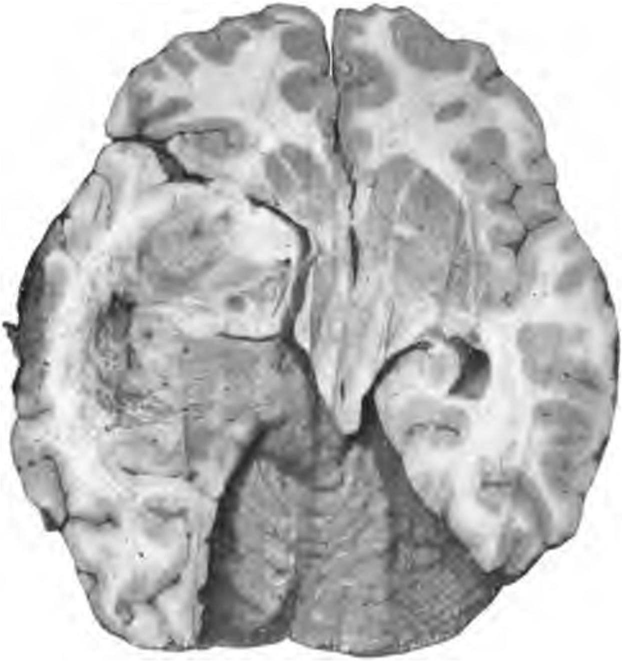

PLATE IV.—Photograph of a section at a level of about ½ of an inch below that shown in Plate III. The tumor fills all the anterior and median portions of the temporal lobe (uncinate and hippocampal convolutions) extending into the middle part of the lobe, including the subcortex of the fourth temporal; it also slightly invades the extreme anterior part of the occipital lobe.—C. K. Mills. (Page 138.)

served was that she said if she looked a certain way to one side she felt as though a spell were coming on.

"Her husband reported that in August, 1906, she began to run down and that her speech was incoherent, with decided aphasia. Whether this was in a technical sense aphasia is doubtful. The patient evidently had some difficulty in remembering words and some slowness and hesitation of speech. Her general health declined so that her weight dropped from 140 to 119 pounds. She came home from her vacation in the summer a physical and almost a mental wreck. She would get out of bed at night unable to tell which way to go to the door. In September, 1906, all medicine was stopped and her old spells began to be more severe and frequent, but she recovered her general tone and her speech. When she was what was termed 'aphasic' she would forget words. She would come into a room, for instance, saying, 'Where are the——,' and after some hesitation she would say, 'Oh, I mean the scissors.' At other times she would substitute wrong words. The patient recovered sufficiently from this so that one would hardly notice any difference between her speech and that of a person who was slightly forgetful of words. She went along about the same until March, 1907. The spells usually occurred in the early morning and her husband would be awakened by a clapping of her lips. One morning on hearing this noise he spoke to and touched the patient, who then went into a general convulsion.

"She was last seen by me in August, 1907. She was at this time very sleepy and drowsy. At the time of her visit to my office she lay down on the sofa and went to sleep. She had at times been taking two or three doses of bromid daily. Little change occurred in her condition until about December 26 or 27, about two months before her death. Between January 22 and the date of her death, February 22, she had perhaps six of the following spells: She would throw up her hands as if in great pain or distress, but could not tell where, except that at the same time she would say that the left side of her body felt numb or asleep or dead. She was not unconscious, but the feelings and condition would last two or three minutes. The morning before her death she had a severe spell. The morning she died she lapsed into unconsciousness and never recovered."

The results of the postmortem examination are in William Spiller's own language:

"The tumor is on the left side of the brain. The left cerebral hemisphere is much larger than the right. In a transverse section at the level of the upper part of the callosum the tumor fills the lateral ventricle above the thalamus, extending to the roof of the ventricle and cutting off the anterior part. At a little lower level, where the anterior and posterior limbs of the internal capsule are well formed, the tumor invades the thalamus, fills the front part of the posterior horn of the lateral ventricle, implicates the extreme anterior part of the median aspect of the occipital lobe, distorts the outer wall of the posterior horn of the lateral ventricle without extending into the temporal lobe, and involves the posterior part of the posterior limb of the internal capsule (Plate II) and a large part of the lenticula.

"At a level about one-half of an inch lower, where the cerebral peduncle is well formed, the tumor fills all of the interior and median portions of the temporal lobe (uncinate and hippocampal convolutions) and the middle part of the lobe, but leaves the occipital lobe intact except in its extreme anterior part, and does not invade the lateral aspect of the temporal lobe or its subcortical white matter (Plate IV).

"It will be noted in the description of the position and extent of the lesion in this case, that it involved the uncinate, hippocampal, and the fourth temporal convolutions and their subcortical white matter, but did not extend further into the temporal lobe. It is probable that the gustatory and olfactory discharges were due to the implication of the cortex in these regions. Of course, it cannot be overlooked that the tumor extended considerably beyond the lower temporal region, invading the anterior part of the occipital lobe and destroying large portions of the lenticula and the thalamus. But with our knowledge of previous cases, and with the experimental evidence regarding the cortical localizations of taste and smell, the findings in this case may be regarded as confirmative of the view that the olfactory and gustatory centers are located in the inferior portion of the temporal lobe.

"So far as the recorded notes show, this patient had no visual phenomena, with the exception that when she looked in a certain direction she felt as if an attack were coming on. No sensations of light, and no hemianopsia appear to have been present. The sensory phenomena may have been due to the involvement of the thalamus and the extreme posterior portion of the internal capsule."

But seven cases of lesions of the uncinate gyrus with necropsy exist in medical literature, and these are here briefly summarized by Mills, six of these being cases collected by Purves Stewart,[1] and the seventh being Worcester's case.[2]

"In the study of cases illustrating the uncinate group of fits, special attention should always be given to the respiratory phenomena. In the case of the patient whose history is recorded in this paper, some peculiarities of respiration in the attacks were noted. At least the patient's appearance was such as to indicate the occurrence of light asphyxia or interference with respiration and cardiac action. Her face would almost invariably turn of an ashen hue. In the severe attacks her face not only became pale as in the light ones, but also became congested."

Hughlings Jackson has especially called attention to states of slight asphyxia which are sometimes observed in the uncinate seizure. In one of the cases recorded by him the patient's face turned blue, or as he quotes the paradoxical expression of some one who observed the attack, "dark pale." He advanced the suggestion that this partial asphyxia and change of color is due to spreading of the discharge lesion in the uncinate gyrus to Spencer's respiratory arrest center, and from this center to the respiratory centers in the oblongata. He speaks of the probable occurrence of writhing movements of the arm in these uncinate respiratory attacks, saying that they are "the consequence of (the physical condition for) suffocation."

Closely resembling, and doubtless belonging to the same group as the case of C. K. Mills, is a case reported by T. Diller[3] under the caption: "A large glioma growing from

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(1) Brain, 1899, XXII, p. 534.
(2) Amer. Jour. Insanity, July, 1887.
(3) Med. Rec., Aug. 28, 1909.

basal ganglia producing symptoms suggestive of a growth in the frontal region." The patient was first seen in 1904, at which time he stated that for two years he had had what he called "smell attacks." These attacks occurred at comparatively short intervals while he was at work in the mill (he was a mill worker), and consisted of a state of mental confusion [dreamy state (?)—ED.], during which he smelt an odor; and sometimes the odor preceded the confused period. [Aura (?)—ED.] He was compelled to sit down during the "smell attacks" and rest for some minutes. He never fell during any of them, nor was he ever unconscious.

A little later he began to suffer from pain in the back of the head, which became very severe. Next his eyesight began to fail. Shortly before Diller first saw him headache had appeared in the frontal as well as the occipital region. In July, 1903, he had a convulsion, and between that time and the following April he had about a dozen more. In these attacks he became unconscious, stiff, cold, pale [ashen (?)—ED.], and breathed heavily. There was apparently very little movement. The patient seemed to sink away; and it is perhaps doubtful whether the attack should be called a convulsion. In April, 1904, there was twitching in the right side of the face and mouth, lasting about half a minute or so, and not being attended with unconsciousness.

Subsequently memory became defective, and he would repeat himself a great deal. His perception seemed slow and dull; he was apathetic and at times irritable. Examination of the eyes showed double optic neuritis. Hearing was impaired in both ears, but more in the right than the left. The sense of smell was very deficient. There were no ocular paralyses and the pupils were normal. Sensation and stereognosis were normal.

Diller says: "As to the location, the symptoms did not appear to point clearly to any one place, but the fact that the smell attacks came early, and that the mental changes appeared to be such as have been described by various authors as occurring in lesions of the frontal lobe, and that there was loss of smell in the right side, made it seem most likely that the tumor was located in the right frontal lobe involving the olfactory nerve by pressure."

Operation was performed over the right frontal lobe, but no tumor was found. The brain was found to be under increased intracranial pressure, however, and the decompression helped the optic neuritis.

Death ensued in August. The postmortem examination showed a tumor springing more especially from the optic thalamus, and with attachments to the former. The tumor grew in an upward direction into the ventricle. "Nowhere did it appear to come in contact with or involve the cortex, unless possibly at the base, which had not yet been incised."

[This case is one which undoubtedly belongs to the uncinate group of cases. It is likely that the cortex at the base involved was that of the uncinate gyrus. The reader is referred to the C. K. Mills case.—Ed.]

Frontal Lobe. A case of frontal brain tumor with the symptomatology pointing to a localization in the posterior fossa is reported by A. Ruckert.[1] The patient was a woman 42 years of age, whose illness began at Whitsuntide, 1907, with transitory headache, vomiting and vertigo, steadily progressing to the point of incapacitating her within six weeks. Later the headaches were alternately in the frontal and occipital regions, and she was tormented with ringing in the ears, especially the right. Finally hearing disappeared from the right ear. Percussion of the head showed an area over the right parietal bone which was very sensitive, and of a different note from that on the left side. Smell on both sides was the same. The visual fields for white and colors were normal, and the eyes could be moved in all directions. The right nasolabial fold was slightly obliterated. Speech was perfect. The spinal column was neither deformed nor tender over the spinous processes. The abdominal reflexes were present, the right knee-jerk lively, and the left reduced. No patellar-clonus, ankle-clonus, Babinski, Oppenheim, or Strümpell phenomenon were present. There were no psychic disturbances. Later, nystagmus was found when the patient looked to the left. By February, 1908, the patient was wholly blind and deaf. On the whole, the subjective symptoms and objective findings seemed to indicate a

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(1) Berliner klin. Woch., July 5, 1909.

tumor of the posterior fossa, but the postmortem examination made March 7, 1908, disclosed a frontal tumor, especially involving the anterior portion of the corpus callosum, in structure a round-cell sarcoma. The complete deafness, first of the right ear, and then of the left, together with the left facial paresis, was not indicative of frontal tumor, and such deafness was not due to middle ear disease as was proved by careful examination.

A similar case was reported by Souques[1] in a man, 40 years of age, suffering with headache both in the frontal and occipital regions, with weakness in the left abdominal muscles, and twitchings in the supply of the left trigeminal nerve. Adventitious ear-sounds followed, first in the left and then in the right ear, and ultimately complete deafness. No middle ear disease existed. In this case, too, a diagnosis of tumor at the cerebello-pontine angle was made, only to find it in the frontal lobe at the autopsy.

Glioma Involving the Pia Mater. A glioma of the brain involving the pia mater is reported by I. Strauss[2] because of the general teaching that gliomata do not infiltrate the meninges. Many instances of gliomata involving the meninges are gathered from the literature, and his own case with postmortem findings is then reported.

Treatment of Cerebral Tumor. In a paper upon the surgical aspects of cerebral decompression C. H. Frazier[3] says: "In the surgery of brain tumor there are but two propositions to consider: the radical operation, implying the removal of the tumor, and the palliative operation, cerebral decompression. In the majority of instances the most we can offer is palliation. But in the case of brain tumor palliation implies much more than it does when applied to other incurable lesions. That is to say, given a patient with an inoperable brain tumor palliation means restoration or preservation of vision, relief from headache and from persistent vomiting.

"Cerebral decompression is called for, as a rule, in inoperable tumors; it may be resorted to, however, as a matter of temporary expediency, when the conditions

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(1) Revue Neurologique, 1904, p. 727.
(2) New York Med. Jour., Dec. 5, 1908.
(3) Jour. Am. Med. Assoc., July 10, 1909.

do not admit of delay and the tumor can not be localized.

"There should be no question about the advisability of operating in comparatively early cases if only to save vision. The propriety of operating in the advanced cases, when the patient is already in deep stupor, may be questioned."

A tumor in the region of the left insula and the first temporal gyrus is reported by Oppenheim and Krause[1], the latter having successfully removed it. Oppenheim saw the patient for the first time November 3, 1907, when she showed aphasia of a sensory and amnesic character. Reading was mechanical, and the subject-matter not understood. In attempting to write, she confused the letters. There was no hemianopia. Double choked disc, worse on the left side, and tenderness on percussion on the left temporal region were found. There was evident paralysis of the right facial at the mouth. A diagnosis was made of tumor in the left cerebral hemisphere, probably in the territory of the left temporal lobe, and radical operation was advised, and performed by Krause. A tumor was found at the anterior inferior end of the fissure of Sylvius, growing from the dura, and compressing the first temporal gyrus and the left insula.

In July, 1909, there suddenly appeared signs of brain compression and symptoms of right-sided paralysis, which compelled a new operation, in which Krause removed a new tumor-mass from the anterior portion of the previous field of operation. In three weeks she was completely recovered. The patient when presented by Oppenheim, October 28, 1908, at the Berlin Medical Society, showed a good degree of intelligence, despite considerable speech defect, since she had resumed all of her household duties. The choked discs, severe headaches, vomiting and vertigo were gone. Reading and writing were still imperfect.

PITUITARY TUMOR.

Pituitary Tumor and Its Surgical Relations forms the title of a paper by Archibald Church.[2] "Regarding the

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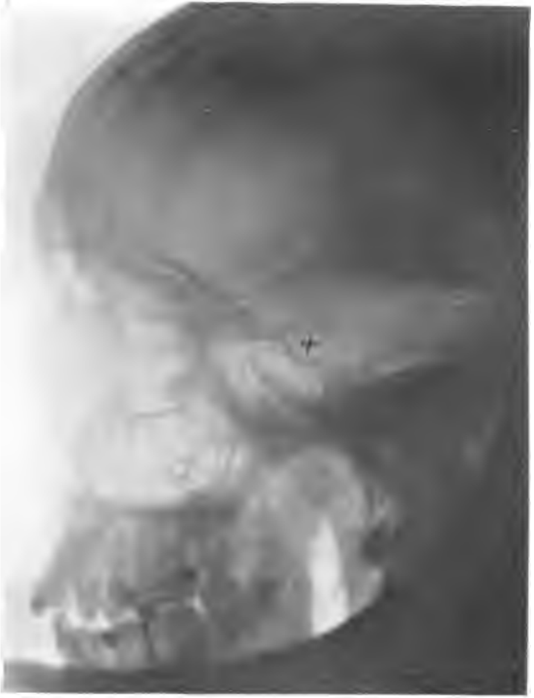
(1) Berliner klin. Woch., Nov. 16, 1908.
(2) Jour. Am. Med. Assoc., July 10, 1909.

symptomatology of hypophysis tumor when unattented by acromegaly, which when present is usually so pronounced as to make the diagnosis self-evident, we are confronted with a series of symptoms that may embrace all those of ordinary brain tumor, but commonly, and probably always at first, are restricted to a few. These, however, are sufficiently definite so that, with the aid of the *x*-ray, the diagnosis may be made with certainty. Hypophysis tumor, when occurring in children, and not producing acromegaly, stunts the growth and the physical and especially the sexual development. The mental qualities may not be materially affected. Owing to the impingement of the tumor or its neck on the optic chiasm, vision is sooner or later impaired, and optic atrophy sooner or later develops. In the early period the atrophy is definitely limited to the nasal half of the disc and this is attended and even preceded by loss of vision in the corresponding half of the field. Bitemporal hemianopsia practically is always due to involvement of the posterior portion of the chiasm, and its detection leaves almost no doubt that it is secondary to disease of the pituitary gland. The rudimentary condition of the genitals or the loss of genital function in those who have once attained it, impotence in men and amenorrhea in women, have a very definite significance in this connection. To this may be added the occasional development of general adiposity and genital dystrophy, of painful adiposity and of acromegaly.

"The *x*-ray is capable of furnishing a picture which clearly outlines the normal sella turcica (Plate V, fig. a), and any enlargement in this bony depression can be positively correlated with an increase in the size of the pituitary body. This was first clearly stated by Oppenheim in 1899."

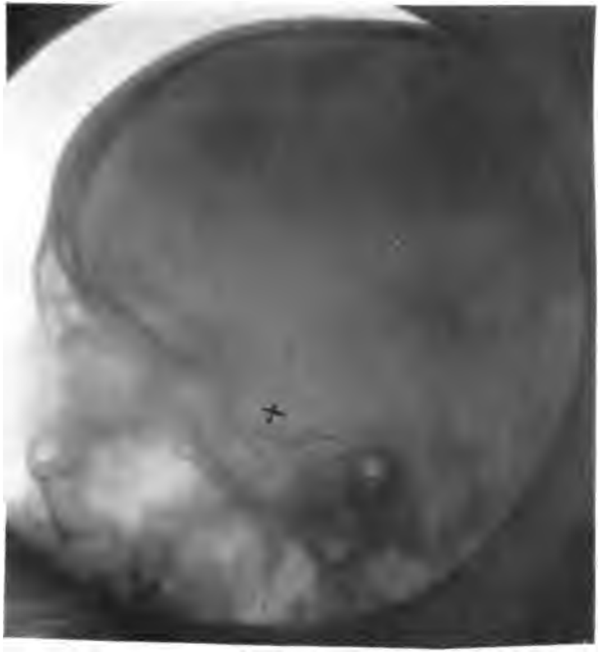
As to surgical intervention, "While the choice of operation is preëminently a surgical question, my own view is that the nasal route is the best. The pituitary tumor is practically extracranial, being located in its very definite bony receptacle below the level of the cranial floor until by abnormal growth it rises into the cranium. As shown by several of my radiograms, the tumor actually descends into the sphenoidal sinus."

Six cases are thoroughly reported by Church, the first one in a woman of apparently good health, 39 years of



a.

Fig. a.—Normal head, showing location and size of the sella turcica, marked by +; much reduced.



b.

Fig. b.—From radiogram in Case 1, much reduced, showing enlarged sella turcica, marked +.

PLATE V.

Illustrating Church's Article, page 144.

(Radiograms Made by Dr. M. Reichmann.)

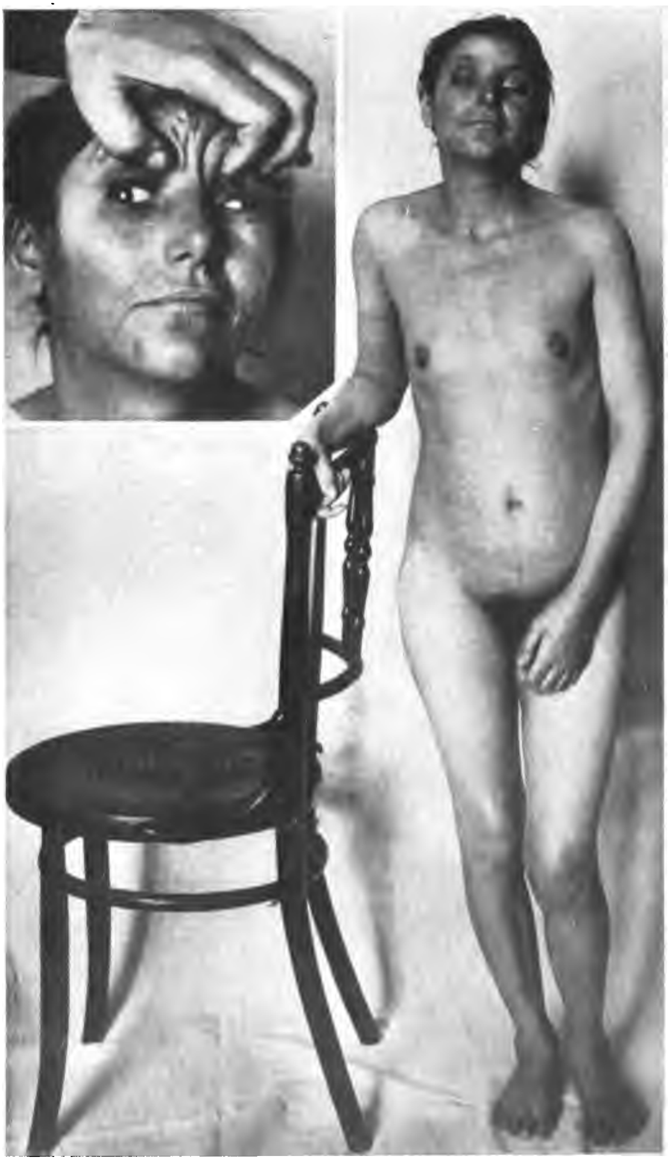


PLATE VI.

The Weber Syndrome. Left hemiplegia and total bilateral ophthalmoplegia.—P. Zosin. (Page 149.)

age. Her menstrual history, double temporal hemianopsia, optic atrophy, and the *x*-ray picture (see Plate V, b) sufficiently established the diagnosis. The second case, a boy of 14 years, showed symptoms suggesting cerebellar disease, and as the family was going abroad the patient was referred to Sir Victor Horsley, who found in a radiogram a pituitary tumor, showing shadows suggesting calcareous plates in the wall of a cyst. Operation was performed and a cystic pituitary body found, containing about an ounce of brown chocolate-like fluid. The brain was found to be definitely hydrocephalic.

Church goes on to say: "The patient made a satisfactory convalescence from the operation and returned to this country, showing a fluctuating state of health. At the site of the operation wound, a pulsating tumor developed, apparently containing cerebrospinal fluid. When this would become tense the boy would become stuporous with intense headache, sometimes with vomiting, and after a few days this condition would subside and he would resume his ordinary activities. The family refused to have even a spinal puncture made, which was advised in the hopes of relieving the intracranial tension, and eventually the boy died about eighteen months after the operation. No postmortem examination was allowed."

In case 3, "the cessation of menstruation, significant visual fields pointing to bitemporal involvement, and the *x*-ray picture made the diagnosis an easy one. Probably this is the first case in which a tumor of the pituitary was diagnosed and operation for its specific removal done in this country."

Church's fourth patient, in a man 36 years of age, a tinsmith, after the diagnosis had been made and operation arranged for, went to Vienna, where the diagnosis was confirmed and operation performed by von Eiselburg, whose fifth case it was, and which is reported in the *Wiener klinische Wochenschrift* (1909, No. 8). The last two cases are similar.

"To these six cases a number could be added in which acromegaly was present. That condition, however, is so self-evident that it is needless to detail instances in this connection. It is much more important that the surgeon, the ophthalmologist and the neurologist should recognize that a pituitary tumor may be present without the association of acromegaly. It is evident from the experience already obtained by the various operators and observers that the removal of the pituitary gland is indicated in both groups of cases. In the first place it furnishes the only possibility of the prevention of blindness, and in the second case seems to hold out some hope of a curative effect for the acromegaly, and in all instances promises the prolongation of life."

CEREBROSPINAL RHINORRHEA.

The escape of large quantities of cerebrospinal fluid continuously, through the nose, is made the subject of a paper by A. Vigouroux,[1] who reports a case with postmortem findings. It is a rare symptom, but few cases existing in the literature. As a rule such escape of fluid is due either to fracture or caries of the sphenoid or ethmoid, or to an enlargement of the holes existing in the cribriform plate of the ethmoid. Such enlargement seems always to be the effect of an excess of pressure of the cerebrospinal fluid acting upon this point. Many names have been proposed for this condition, such as cerebrospinal rhinorrhea, craniorrhea, and cerebrospinal catarrh. A case reported by G. Noccioli in 1904 was caused by a pistol-ball. Some of the cases reported have been due to caries of the bones, some to congenital hydrocephalus.

Freudenthal[2] reported a case in a woman 50 years of age suffering from excessive frontal pain just above the nose. Her attacks ceased when the cerebrospinal fluid began to flow from her left nostril. The sense of smell was lost.

Vigoroux's case is grouped by him with three others in the literature, the cases of Nothnagel, of Wollenberg, and Adolf Meyer.[3] It showed the escape of cerebrospinal fluid through the nose for a space of four years. It came on when the patient was 28 years of age, after a violent headache. For a year about 800 c. c. escaped daily; then convulsive accidents and mental changes appeared

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(1) La Presse Méd., May 15, 1909.
(2) Jour. Am. Med. Assoc., Feb. 26, 1894.
(3) N. Y. Neurol. Soc., Jan. 3, 1905.

whenever the escape was temporarily interrupted. The autopsy showed a voluminous papilloma of the choroid plexus of the fourth ventricle, an enlargement of the holes in the cribriform plate of the ethmoid, communicating with the nasal fossæ, an enormous dilatation of the pituitary fossa, marked dilatation of the lateral ventricles, the third ventricle, and the Aqueduct of Sylvius, and an adhesion of the internal face of the frontal lobes to each side of the crista galli. The symptom is explained by the author as due to papillomata of the choroid plexus, the dilatation of the holes in the cribriform plate, and the intermittency as due to the occasional exacerbations of a meningitis sealing temporarily the orifices of exit to the fluid.

In all cases reported with postmortems in the literature the cause of the escape of the fluid is either (1) trauma, or (2) caries of the ethmoid, or (3) a very marked hydrocephalus from any source producing great pressure on the cribriform plate. The symptoms are those of hypertension of cerebrospinal fluid and of cerebral tumor. Headache is always present, vertigo, hebetude, and epileptiform seizures follow, with disturbances of vision and the loss of the sense of smell. The influence of the hypertension of the fluid upon the intensity of the symptoms is indicated by the remission of the latter when the fluid runs freely, and the exacerbation when the flow is prevented. The fluid does not burst through the dura mater directly, but dissects away the sheaths of the dura mater about the olfactory filaments as they pass through the cribriform plate, and exudes between the nerve-sheath proper and the dura mater.

OPTIC THALAMUS.

Optic Thalamus Syndrome. A case of tumor of the optic thalamus from the service of Mingazzini at Rome is reported by A. Piazza.[1] In a young man 22 years of age without antecedent illness there suddenly appeared in the midst of excellent health diffuse headache, vomiting, diplopia and amblyopia. Objectively the only demonstrable symptom was paresis of the left rectus externus and choked discs. Later both internal recti became paretic, and peripapillary hemorrhages appeared in the fundi. Hearing became reduced on both sides, and the gait uncertain. Later the diplopia and choked discs improved, but optic atrophy and deafness increased. Headache and vomiting continued without abating and the patient sank into a state of marasmus and mental dulness, with involuntary passage of urine and feces. Generalized convulsions then followed. While the patient was in this state a kind of "cracked-pot" sound could be obtained on percussing the skull over the right parietal region. Death ensued about ten months after the onset.

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(1) Berliner klin. Woch., Aug. 30, 1909.

The autopsy showed a sarcoma springing from the third ventricle, extending over the corpora quadrigemina and the right optic thalamus. It had destroyed the mesial and inner portion of the lateral nuclei of the optic thalamus, had compressed the nucleus ruber of the midbrain, had caused degeneration of the mesial four-fifths of the pes pedunculi, and had spread to the right gyrus hippocampus. The ventricles were much dilated.

In this case a localizing diagnosis was practically impossible. Indeed the involvement of the sixth and eighth cranial nerves might have suggested a specific lesion. Percussion was of no diagnostic value. Other cases of tumor of the third ventricle have been reported by Mott and Barrat (3 cases), Henneberg and Williamson (one each), and are here reviewed. Tumors involving the optic thalamus have been reported by Remak, Calonzi, and Glasow, these cases being here briefly summarized; and reference is also made to Roussy's Thalamic Syndrome (see Practical Medicine Series, 1908, Vol. X, pp. 118-122).

Not one of the symptoms laid down by Roussy was present in this case, and Piazza is skeptical concerning the optic thalamic syndrome.

Four cases showing the group of symptoms collated by Déjerine and Roussy as comprising the thalamic syndrome are reported by I. H. Coriat,[1] but since no postmortem findings accompany the cases, they are here merely mentioned.

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(1) Jour. Nerv. and Ment. Dis., August, 1909.

MIDBRAIN.

THE WEBER SYNDROME.

A case of left-sided hemiplegia with total bilateral ophthalmoplegia is reported by P. Zosin[1] in a woman 31 years of age, without specific history or any other etiological suggestion. After a hard day's work, the evening meal being over and the children having been put to bed, she herself retired, apparently in perfect health, without any previous headache or vertigo. Shortly after midnight her husband and children were awakened by her stertorous breathing, and they feared she was dying. All that the patient remembered, after unconsciousness for a week, was a frightful dream. When she regained consciousness she could not get out of bed, could not raise her eyelids, and could not move her left hand or leg. Speech was absolutely lost for three weeks, when it returned almost completely. Plate VI shows her present condition quite admirably. Very striking is the bilateral ptosis, the right facial paresis, and the position of the bulbs. The pupils were absolutely inactive (*ophthalmoplegia interna*). Subjectively there was also a left-sided hypesthesia. Such a symptom complex can scarcely be due to anything else than a lesion in the right side and central portion of the midbrain, involving as it does both the right and left oculomotor nuclei, the right pyramidal tract, and the right fillet in the midbrain. The lesion must have been a hemorrhage or an embolus.

Three cases of superior alternate hemiplegia, of the Gubler-Weber type, are reported by S. E. Jelliffe.[2] After calling attention to this form of crossed hemiplegia first described by Gubler but later and more thoroughly by Weber, Jelliffe mentions a *first* case, antedating those of Gubler and Weber, reported in 1858 by Luton, summarizing it in detail. Jelliffe's first case occurred in a laborer 44 years of age, who, after a stroke, showed complete left-sided hemiplegia, with ptosis of the right eyelid, and dilated right pupil, with external squint of the right eye.

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(1) Nouv. Icon. de la Salpêtrière, March-April, 1909.
(2) Interstate Med. Jour., September, 1908.

The second nerve showed some defect on the right side, but none on the left, and there were neither diplopia nor hemianopia. The third nerve showed marked involvement on the right side, there being ptosis and paralysis of all the muscles except the superior oblique and external rectus. The left eyeball could be moved normally. The second and third cases are almost counterparts of the first.

As to the question of hemorrhage or softening as a cause of the Weber syndrome, it may be said that softening in the cerebral peduncle due to emboli is rare; but softening due to arteritis, especially of syphilitic origin, may occur. In two of Jelliffe's three cases hemorrhage of the artery supplying the third nerve nucleus was the cause of the syndrome. The ophthalmoplegia is to be looked upon as the main or primary symptom, and the hemiplegia is to be interpreted as a secondary and complicating symptom.

POLIOENCEPHALITIS SYPHILITICA.

Polioencephalitis syphilitica occurs, but it is very rare. A case is reported by Dieulafoy[1] at great length, and exceedingly ably and convincingly. The patient, a young man 29 years of age, showed a double ptosis, with a complete bilateral ophthalmoplegia, accompanied also by bulbar symptoms. His eyeballs were absolutely immovable and fixed in a position of external strabismus. Thus every external branch of the third nerves and both fourth nerves were paralyzed, the sixth nerves alone being exempt. In short, the patient showed the syndrome named by Brunner in 1850, external ophthalmoplegia. But he also showed what Hutchinson in 1879 named internal ophthalmoplegia, both the sphincter iridis and the ciliary muscles being likewise bilaterally paralyzed.

The onset of this ophthalmoplegia had been very gradual, extending over eight months, beginning in the left levator palpebræ superioris, three weeks later involving the right, and finally, little by little, all of the external and internal muscles, except the external recti. Examination with the ophthalmoscope showed nothing. There was, however, the scar of an old keratitis on the left cornea. Such ophthalmoplegia might be tabetic, diabetic, or

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(1) La Presse Méd., Nov. 11, 1908.

syphilitic, but no signs of tabes or of diabetes could be discovered. No basal lesion could produce the symptoms, which must have depended upon a central nuclear lesion. Basilar lesions cannot produce an external and internal ophthalmoplegia of *gradual* and, as it were, of *selective* onset. Such an ophthalmoplegia deserves the appellation polioencephalitis anterior or superior.

In this case, however, there were bulbar signs, indicating the existence of a polioencephalitis posterior or inferior. Before the patient entered the hospital there was a very intense polyuria and polydipsia, suggesting a bulbar lesion. The polydipsia appeared abruptly, without cause, and led to a polyuria of from 12 to 15 litres per 24 hours. There was no glycosuria, phosphaturia, or other indication of anything beyond a simple polyuria. About the same time swallowing became very painful, so that the patient ate only soup and milk. A syphilitic gumma found on the right pillar of the fauces seemed to account for the dysphagia, which disappeared when the gumma healed. But the polydipsia and polyuria persisted, and the patient became sleepless and very emaciated by reason of anorexia. Subsequently other bulbar symptoms set in with intensity. The soft palate became paralyzed, especially on the left side; the voice became nasal; and fluid and food came through the nose when the patient attempted to swallow. Feeding by the stomach tube had to be adopted. Then there was a tendency to syncope, vomiting, and vertigo, indicating involvement of the nucleus of Deiters in the pons. Incontinence of the sphincters, with escape of the urine and feces followed. The tongue deviated very markedly to the right, and the right side was notably atrophic, indicating a lesion of the hypoglossal nucleus. The labial commissure deviated to the left when the patient smiled or showed his teeth, the right side of the face being paretic. Finally there was a slight atrophy of the right sterno-cleido-mastoid, and a very marked atrophy of the upper half (clavicular portion) of the right trapezius, indicating a lesion of the right spinal accessory nucleus. Hence the justification of the diagnosis of polioencephalitis posterior and anterior in this case.

It was from the first felt to be a purely syphilitic case, but previous antisyphilitic treatment had availed little, the symptoms developing in spite of the treatment. Dieulafoy gave him 48 injections of from 1 to 2.5 centigrams of the biniodid of mercury, with but short interruptions between them. Gradually the headache disappeared, the voice was restored, swallowing became possible, the eyeballs became movable, the upper lids became active, the right side of the tongue filled out, the tongue was protruded in a straight line, and the facial assymetry disappeared. A year later the trapezius had filled, out, and so had the sterno-cleido-mastoid.

When the symptoms present are due to gummata the effect of treatment may be marvelous. When the symptoms are due to specifically diseased blood-vessels brilliant cures are not to be expected. In this case the polioencephalitis appeared six years after the chancre, and was undoubtedly gummatous in origin. It is not the only case of specific polioencephalitis reported and cured, Achard[1] having reported a case. Dieulafoy concludes as follows:

"(1) There is a syphilitic polioencephalitis. This polioencephalitis may betray itself by ophthalmoplegia and bulbar symptoms.

"(2) The bulbar symptoms and ophthalmoplegia may be associated or isolated, but from the moment that it is found to be syphilitic it is to be treated specifically; but such treatment to be effective must be prompt, long continued and of sufficient intensity."

CEREBELLUM.

CEREBELLAR TUMOR.

Differential Diagnosis. This subject forms the title of a paper by Ernest Jones, read before the annual meeting of the Ontario Medical Association, June 1, 1909,[2] the article being based upon some twenty personal cases. Of the general symptoms, headache is very pronounced and is invariably confined to the sagittal plane. Optic neuritis is early, rapid in onset, and intense in character. Instead of the ordinary vertigo of a cerebral tumor there is a peculiar sense of lateral rotation, things seeming to turn from the side of the lesion. The attitude is often a forced one, the head being held in a position of lateral flexion, the ear on the side of the lesion being approximated to the corresponding shoulder. (See Practical Medicine Series, Vol. X, 1904, p. 161.) Station is changed, the patient resting on a broad base, the weight being chiefly supported on the side opposite the lesion. The gait has two special features: either the patient reels as if drunk, or he walks in a curve, the concavity of which is on the side of the lesion. [*He walks around his lesion.*—ED.]

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(1) La Bulletin Méd., April 11, 1906.
(2) Boston Med. and Surg. Jour., Aug. 26, 1909.

The ataxia is peculiar in being of the dynamic variety, thus differing from static ataxia. It is not due to any loss of the "muscle sense." It is always more marked in the upper than in the lower limb, and is usually confined to the side of the lesion. The carrying out of a complex movement frequently shows a defect that Babinski has termed "cerebellar asynergy." For instance, if the patient is told to straighten a lower limb that is flexed at both hip and knee-joints he will extend first the leg and then the thigh, and not both simultaneously, as in the normal. Again, he is unable to carry out any rapidly reciprocating movements, a symptom known as dysdiadokokinesia.[1]

"A cardinal side of cerebellar disease is *hemiparesis.* This paresis is distinguished by being more pronounced in the trunk than in the limbs, and in the upper than in the lower limb; by never affecting the face; by being of a flaccid and not a spastic type, and by being unaccompanied by pain or any other sensory disturbance. The *hypotonia* that accompanies this paresis is a highly important differential sign. It is shown by the diminished resistance to passive movements, by the flaccid feel of the muscles and the greater mobility of the joints.

"Of the *eye-symptoms,* three are of especial importance: nystagmus, sixth nerve weakness and skew deviation. Cerebellar nystagmus is characterized by being most marked when the patient looks towards the side of the lesion. It is practically always lateral in type. The

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(1) See Practical Medicine Series, 1903, Vol. X, p. 162.

paresis of the sixth nerve is an exceedingly frequent symptom; there is commonly also a dissociated paresis of the opposite internal rectus muscle so that it is weak in lateral movement of the two eyes, though not in convergence. There are several kinds of skew deviation of the eyes. The commonest is the Magendie type, in which the homolateral eye looks downwards and inwards and the contralateral eye outwards and slightly upwards. It is usually a temporary phenomenon and, therefore, has carefully to be watched for."

Differential Diagnosis from Other Tumors. Of *supratentorial* tumors, those that give rise to the greatest difficulty are tumors of the frontal lobe, of the optic thalamus, and of the corpora quadrigemina. "A *parietal* tumor may occasionally be a source of embarrassment in diagnosis. It gives rise to a lack of dexterity in the limbs, which, however, is due to an astereognosis or sometimes to sensory asymbole, and so is quite different from cerebellar ataxia. When the tumor extends far back in the parietal lobe it may produce conjugate deviation of the eyes, but in such a case there will probably be present a contralateral homonymous hemianopsia or else a mind-blindness for objects, together with evidence of visual aphasia.

"*Frontal* tumors give rise to many symptoms resembling those of cerebellar tumors. In both cases there may be present nystagmus, conjugate deviation of the eyes, speech disturbances, unilateral tremor of the limbs, and even the so-called cerebellar attitude of the head. The conjugate deviation of the eyes in frontal tumor is, however, an irritative phenomenon, not, as in cerebellar tumor, a paralytic one, and the two can thus be readily distinguished. The speech disturbance is of a different kind, being aphemic and not dysarthric. The tremor has a different character, being very fine and rapid, and there is never present the typical cerebellar ataxia. Hemiparesis when present is on the opposite side and is of the cerebral type, with spasticity and the characteristic changes in the reflexes. Symptoms, such as anosmia, apraxia, bitemporal hemianopsia, agraphia and motor aphasia, may occur that are never found with cerebellar tumor.

"Tumors of the *optic thalamus* can very easily be mistaken for cerebellar tumors. The hemiparesis is slight, and still more striking is the fact that it is hypotonic. Vertigo and the cerebellar attitude of the head may occur, and further, the involuntary irregular movements that constitute a cardinal sign of thalamic lesions are sometimes very hard to distinguish from cerebellar ataxia. However, with tumors of the thalamus there are always pronounced sensory changes, particularly loss of deep sensation, and marked irritative symptoms, such as pain and paresthesia. Implication of the third nerve nucleus is common, most often manifesting itself as a bilateral mydriasis. In all the cases of thalamic disease so far reported, there has been some implication, however slight, of the motor part of the internal capsule.

"It is sometimes impossible to distinguish a tumor of the *corpora quadrigemina* from one of the cerebellum. A few points of value are that the deafness is an early symptom and not a late one, as in cerebellar disease; that it is on the side opposite to the tumor; that the affection of the third nerve, causing most often external ophthalmoplegia, is one of the first signs that hemianopia may occur, and that the ataxia and tremor are frequently bilateral. Paresis when present is frequently bilateral, and is always spastic, being due to implication of the pyramidal tracts.

"The difficulties of distinguishing cerebellar tumors from other *subtentorial* tumors are even greater than in the case of supratentorial ones, and here accuracy in localization is of vital importance because on it depends the operability of the case. Tumors of the *pons* and *medulla* are relatively easy to distinguish. The optic neuritis is late in appearing and the general symptoms are not pronounced. Vertigo and ataxia may occur, but not of the cerebellar variety. The sphincters are frequently affected, and there are often vasomotor and respiratory disturbances. The paralysis may be on one or both sides of the body, but is always spastic and is accompanied by evidences of affection of the pyramidal tract, such as Babinski's plantar sign, etc."

Differential Diagnosis from Hydrocephalus. Hydrocephalus frequently produces cerebellar symptoms, and such a case, associated with syringomyelia, with postmortem findings, is reported by J. H. W. Rhein.[1] The patient, a boy 8 years old, about October 23, 1907, began to have headaches and vomiting. The head was found enlarged. The eyes, examined by W. C. Posey, showed nothing. Shortly after this there developed a spastic gait, but otherwise nothing of importance was noted until two years later, November 29, 1907, when the child again returned with a history of vomiting, attacks of frontal headache, and general tremor. Station was poor, and the knee-jerks were increased. There was a history of a general convulsion three years previously, and of several attacks, in which the patient fell, during the past year.

Examination January 8, 1908, showed the following: "There was a coarse, irregular tremor of both hands on voluntary effort. The eyes were prominent, and the palpebral fissures unusually wide, showing the cornea above the pupil. The tongue was protruded straight, and there was no facial palsy. There was no stiffness of the neck, nor apparent tenderness in that region. The knee-jerks were increased, and equally so. There was no ankle-clonus, but the Babinski phenomenon was present on the left side. Kernig's sign was also present. The pain sense was generally preserved. Moreover, there were no local atrophies to attract attention to the possibility of the existence of syringomyelia. The patient staggered when walking, and seemed to be weaker in the left leg than in the right. The sphincters of the bladder and rectum were incontinent.

"January 12, 1908, the headache was very severe; the head was retracted and the neck stiff. Any movement of the head increased the pain, which appeared to be general. At this time the knee-jerks were slightly diminished, and there was a tendency to fall to the left, with the feet together and the eyes closed. The patient died suddenly."

Autopsy. When the brain was removed 6 to 8 ounces of cerebrospinal fluid escaped, and the very much distended brain collapsed to a certain extent. What appeared to be two cysts were observed at the cerebello-pontine angles, distended with fluid, which escaped when the brain was removed. (See Plate VII, a. A and B.) Both lateral ventricles were very much distended. (See Plate VII, b.)

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(1) Jour. Amer. Med. Assoc., Dec. 5, 1908.

The third ventricle was also greatly enlarged, pushing the tissues of the interpeduncular space downward. The Aqueduct of Sylvius was increased in size, as well as the fourth ventricle, especially the cerebellar portion, and the lateral processes. These were continuous on each side, with the cysts situated at the cerebello-pontine angles. The foramina of Magendie and Luschka could not be found.

The spinal cord was the seat of a syringomyelia which extended from the eighth thoracic to about the second cervical segment.

A similar case is reported by Bullard and Thomas[1] in a boy 6½ years old, presenting symptoms of a cerebellar lesion. Later there developed paraplegia and incontinence of urine and feces. The autopsy revealed the presence of hydrocephalus and syringomyelia, the latter not having been suspected during life.

Clinical Cases. An unusual case of cerebellar tumor is reported by J. E. Dale[2] in a boy 16 years of age, who, on July 7, 1908, was suddenly seized with vomiting during the night, and fainting on one trip to the bathroom. There was no headache, and nothing on physical examination. During September there was obstinate constipation, and more vomiting, from which he recovered. On September 26 the patient came to the office, saying: "I have to make myself breathe, and I feel as if I could not do it." Examination of the heart and lungs was negative. At 9 p. m. vomiting began again. The next day his condition was unchanged; he still complained of difficulty in breathing; he was very nervous, and there was occasional slight vomiting. On September 28, at 1:30 p. m., the pulse was 78, the respirations 20, and to all appearances normal, but the patient complained bitterly of difficulty in breathing. "At 3 p. m. breathing suddenly became more shallow and slower, the patient became unconscious, the left eye was closed, the pupil contracted, the right eye open, the pupil dilated. The mouth was drawn to the left. Marked cyanosis rapidly developed, becoming more and more intense. There was no further movement except slight twitching of the left side of the face. The pulse remained

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(1) Am. Jour. Med. Sciences, 1899, p, 265.
(2) Med. Rec., Aug. 7, 1909.

normal up to a few minutes before death, which occurred one hour after the first appearance of paralytic symptoms.

"The brain was removed and sectioned. Attached to the roof of the fourth ventricle, slightly to the right of the median line and directly over a point in the floor slightly posterior to the center, was a growth about one centimeter in diameter, somewhat pedunculated, the surface rough, about the color of the surrounding tissue, macroscopically resembling a villous wart. In the substance of the cerebellum about the base of the tumor for a distance of two centimeters or more the outline of the arbor vitæ was blurred and indistinct."

Three cases of cerebellar tumor, with operation in two cases and recovery in one, are reported by Wharton Sinkler.[1] Operation varies widely as to the beneficial results obtained. For example, Sachs says that out of twenty-three of his patients, not one was cured in seven operated. Frazier's collection of 116 cases made in 1904 showed recovery in 15%; and Starr, in 1903, gave as a result of operation in 58 cases of tumors of the cerebellum, recovery in 16. In these cases of Sinkler two were operated upon and one recovered. The growth in this case was a cyst; the contents were evacuated, and the wound closed, but vision was practically lost because of delay in the operation. In the second case the patient had shown no signs of brain disease until nine weeks before seen by Sinkler. Her symptoms progressed so rapidly and vision had begun to fail to such an extent that, although there were no definite localizing symptoms, a decompressive operation was deemed advisable. There was paralysis of the right sixth nerve, and this fact led to the selection of a point over the right cerebellar hemisphere for trephining. No growth was found at the operation, but at the autopsy made, thirteen days later, a tuberculous tumor was found in the anterior part of the right hemisphere, infringing upon the medulla, and making a marked depression in its right side. In the left hemisphere were two smaller tumors. "The fact of there being a growth in each hemisphere would account for the bilateral symptoms presented by the patient during life. In this case, although

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(1) Jour. Am. Med. Assoc., Sept. 26, 1908.

the swelling of the discs subsided in a remarkable manner after the operation, the vision continued to fail." The third case did not live to be operated upon.

A case of cerebellar tumor and its successful removal is reported by T. Diller and O. C. Gaub.[1] The patient, a young man 20 years of age, suffering with headaches, showed a drawing of the head forward and to the left. There was marked vertigo and right-sided sweating. Gait was weak and uncertain, upon a broad base. Diagnosis of the side of the cerebellum was not made, hence both lobes were exposed, Cushing's method being followed. A tumor the size of a hickory nut was enucleated from the anterior surface of the right lobe. Recovery followed.

A case of tumor of the cerebellum is reported by Gierlich[2] in a child 7 years of age, presenting headache, vertigo, vomiting, cerebellar ataxia, choked discs, nystagmus, spasms of the lower extremities and intention-tremor. In the right arm, nine months after headache began, adiadokokinesia was plainly observed, but sensation, sense of position, and the stereognostic sense were intact. The patellar and Achilles-jerks were exaggerated in both sides, and there was ankle-clonus, the Babinski toe-sign and the dorsal foot-reflex of Bechterew-Mendel. The corneal reflex was reduced and at times absent. Complete blindness threatening, an operation was performed, the tumor being supposed to be in the worm or right hemisphere. Tumor masses were found in the *incisura posterior* springing from the lower surface of the worm, in structure a round-celled sarcoma. In 48 hours the child died. The autopsy more completely revealed the tumor and its location.

Important points are the intensity of the cerebellar ataxia which confined the patient to bed, doubtless caused by the destruction of fibers from Deiter's nucleus to the worm. No tendency to fall backwards existed, despite the teaching that lesions of the posterior end of the worm provoke such a tendency. Another point is the coarse intention-tremor, which by Stewart is ascribed to hydrocephalus, present in this case. The adiadokokinesia of the right forearm was doubtless due to pressure upon the right cerebellar hemisphere.

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(1) Jour. Am. Med. Assoc., July 31, 1909.
(2) Deutsche med. Woch., Oct. 15, 1908.

TUMOR OF THE CEREBELLO-PONTINE ANGLE.

A tumor of the cerebello-pontine angle of a peculiar course and symptomatology is reported by Gierlich.[1] The patient, a woman 28 years of age, was taken ill with headache, a tendency to vertigo, and failure of vision. Examination of the fundi showed beginning optic atrophy without disturbance of motility of the bulbs or of the pupillary reflexes. Later the gait became uncertain, especially on suddenly turning. Ataxia of the cerebellar type followed, and the sense of smell was lost on both sides. The sense of taste on the right side of the tongue for sweet, sour, salt and bitter was also limited. Subsequently blindness became very pronounced, and there developed a paresis of the left side of the body, and the lower part of the facial nerve supply. A little over three years after the first symptom there suddenly appeared complete inability to swallow, limitation of speech, and dysarthria. Two months before her death nystagmus appeared when she looked to the right, the occipital headache was right-sided, and she was unable to stand. From this time on feeding was wholly by the stomach tube. Other signs present were left ankle-clonus, Babinski's sign, the Oppenheim and Bechterew-Mendel reflexes, hypotonicity of the left leg, and absence of the right corneal and conjunctival reflexes.

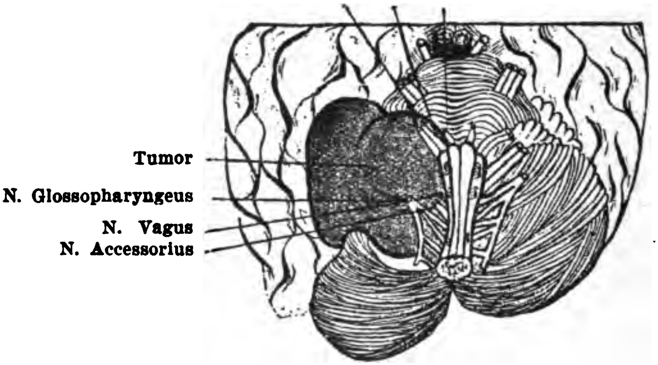

Fig. 12.—Cerebello-pontine tumor.—Gierlich.

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[1] Deutsche med. Woch., Oct. 15, 1908.

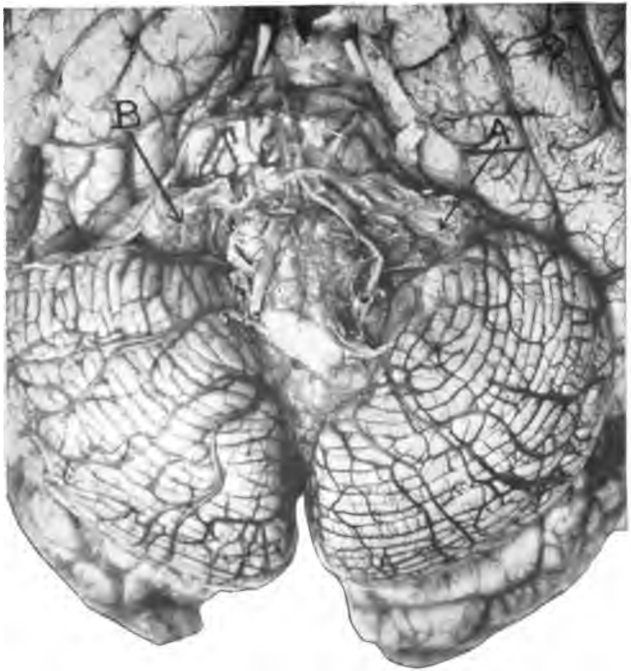

Fig. a.—View of the hydrocephalic brain, showing two cysts, A and B.—J. H. W. Rhein. (Page 156.)

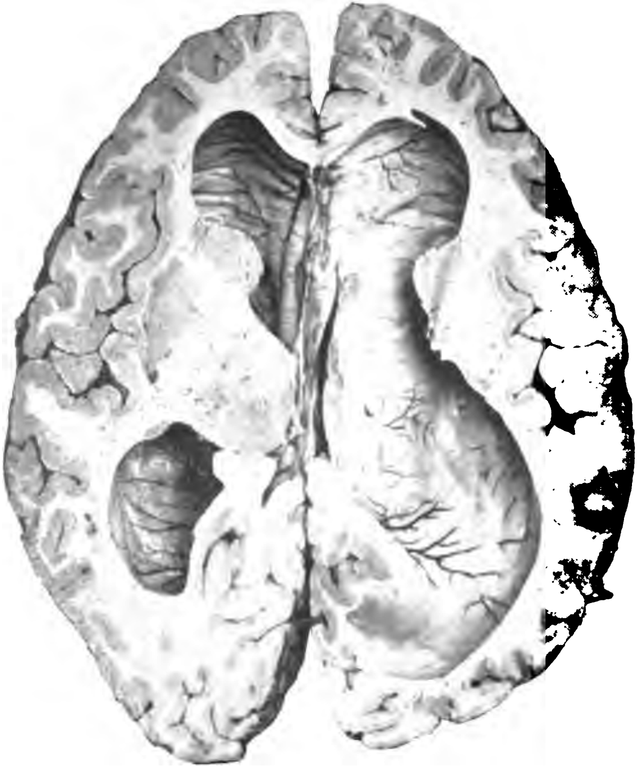

Fig. b.—Cross-section of the hydrocephalic brain showing distension of the lateral ventricles.—J. H. W. Rhein. (Page 156.)

PLATE VII.

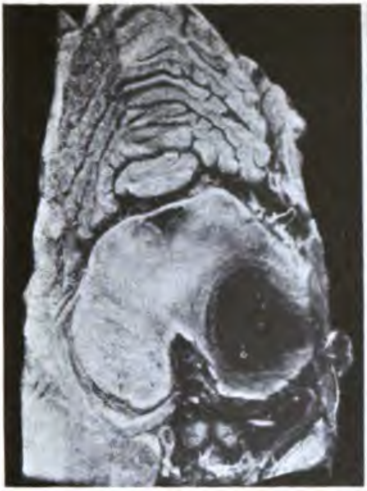

Fig. a.—Section through the cerebellum and pons Varolii at the level of the crura cerebri and 3rd nerve nuclei.

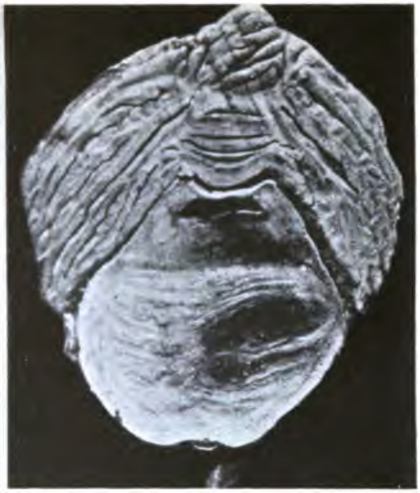

Fig. b.—Section through the cerebellum and pons Varolii at the greatest thickness of the pons. The hemorrhage has infiltrated the tissue of the pons; strands of nervous tissue are seen passing between masses of hemorrhage.

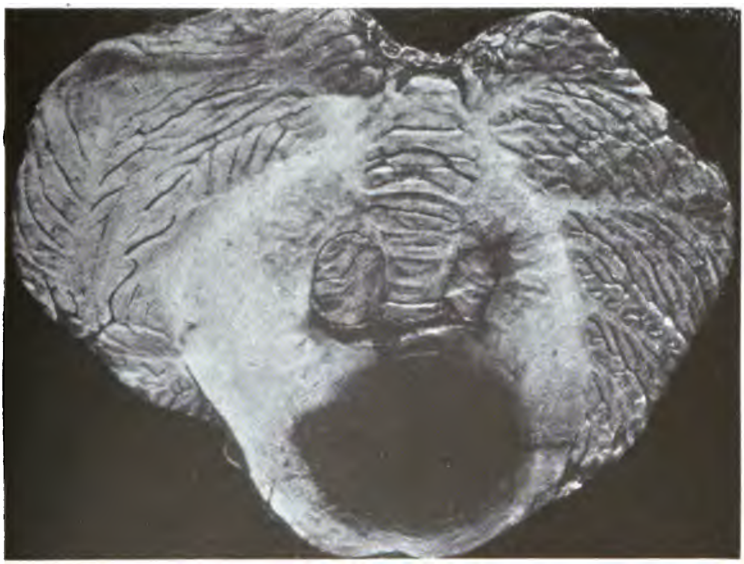

Fig. c.—Section through the cerebellum and pons Varolii at the level of the middle crura of the cerebellum. The hemorrhage was most extensive at this level, and, had, practically speaking, destroyed almost the whole of the pontine tissue.

PLATE VIII.

Pontine Hemorrhage.—Bramwell. (Page 163.)

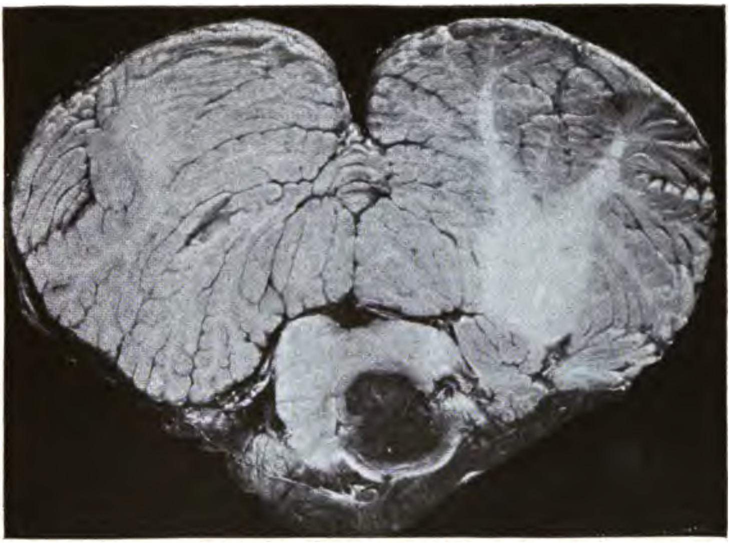

Fig. c.—Section through the cerebellum and medulla oblongata at its upper end.

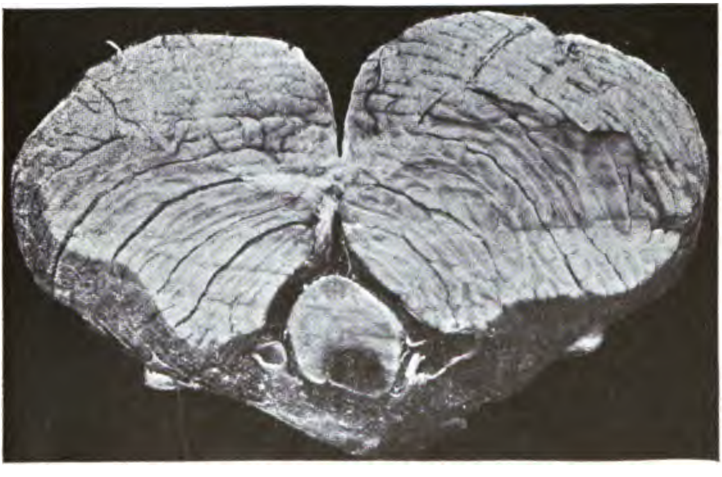

Fig. d.—Section through the cerebellum and medulla oblongata, just above the decussation of the pyramids.

PLATE IX.

Pontine Hemorrhage.—Bramwell. (Page 163.)

Postmortem examination disclosed a fibrosarcoma, starting from the perineurium of the glossopharyngeal nerve, and filling the right cerebello-pontine angle, shown in Fig. 12. The case clinically was peculiar. First, optic atrophy was found without choked discs; secondly, there was loss of the sense of smell. These facts suggested a tumor in the anterior fossa. Later cerebellar ataxia, loss of the taste sense, a crossed hemiplegia, and right-sided auditory and trigeminal symptoms, indicated a tumor in the posterior fossa. Hence multiple tumor was diagnosed. The postmortem showed that a very pronounced hydrocephalus internus had caused the loss of smell by pressure on the olfactory bulbs, and that the pressure of the distorted infundibulum upon the optic nerves had caused the optic atrophy.

PONS AND MEDULLA.

Agenesis of the Facial Nucleus.

Unilateral facial paralysis and bilateral external ophthalmoplegia, congenital in origin, are reported by Babonneix and Harvier[1] in an infant one year of age, born at full term of healthy parents. Congenital paralyses of cranial nerves, among them the seventh, are not rare, cases having been reported by Cabannes, Marfan and Delille, Souques and Heller, and others. Of these cases there are two groups, one of congenital agenesis of the petrous portion of the temporal bone, the other being agenesis of the nucleus of the seventh. The latter are apt to be bilateral (congenital facial diplegia), but may be isolated, or associated with an external ophthalmoplegia as in this case and others. The lack of response on the part of the seventh to electricity here reported speaks in favor of the agenesis of its nucleus, and it is likely that the other nuclei controlling the eye-movement here lost, are also agenetic. Examination of the fundus showed an atrophic choroiditis and an absence of retinal pigment, and these facts favor the congenital agenesis theory in the case.

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(1) Gaz. des Hôp., Nov. 5, 1908.

PONTINE HEMORRHAGE.

A remarkable case of hemorrhage into the pons and medulla is reported by Byrom Bramwell[1] as being unique in his experience. After a small primary hemorrhage which occurred three weeks previously, an extensive hemorrhage followed slowly and gradually, lasting about a week, yet producing no unconsciousness up to the time of death. The patient, a healthy man, 31 years of age, never previously ill, got up on June 17, 1908, feeling dizzy, seeing double, and feeling weak in his left arm and leg. He started to work, but reeled and staggered like a drunken man, gave up the attempt and went back to bed. Seen by Bramwell four days later, he was found to have a temperature of 98.8° F., a pulse of 88, a respiratory rate of 16, and a blood-pressure of 150 mm. The urine was negative, the heart normal. The left leg and arm were weak and numb, and reduced to the sense of pain. There was, however, no marked anesthesia or analgesia. The right external rectus muscle was paralyzed and some nystagmoid movements were seen in the left eye, when conjugate movement to the right was attempted. The pupils and optic discs were normal. The sudden onset suggested a vascular lesion, but its nature was doubtful, and the conditions of the heart and kidneys did not favor it. All things taken into account, Bramwell was disposed to think that the most likely lesion was a small vascular glioma of the pons at the level of the sixth nerve nucleus, which up to the time of the occurrence of a presumable hemorrhage from it had never produced symptoms.

For a fortnight the patient remained in an unchanged condition. On July 8 he stated that he felt weak all over, and complained of right-sided frontal headache. On July 9 the weakness in the right arm was distinctly worse, and there was some difficulty in articulation, due to the fact that he could not properly move his tongue in his mouth. There was also some deviation of the tongue to the right, and some difficulty in swallowing. On July 11 the right frontal headache became worse, and he vomited. The following day there was retention of urine and more vomiting, and hearing in the right ear (hitherto normal) was

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(1) Edinburgh Med. Jour., February, 1909.

found to be considerably impaired. July 13 the right external rectus was found to be completely paralyzed and there was defective conjugate movement of the eyes to the right. There was also some ptosis. The pupils were equal and active, and the disc normal. Ankle-clonus and an exaggerated knee-jerk and Achilles-jerk in the left side were also found. The left abdominal reflex was absent, and the right much reduced.

On July 14, exactly a month after the onset of the disease, the patient died, being quite conscious until shortly before death. The autopsy showed findings as in Plate VIII, a, b, and c, and Plate IX, a and b. The hemorrhage extended from the top of the pons and right crus cerebri above to the bottom of the medulla below as far as the decussation of the pyramids. At its upper and lower limits the hemorrhage is chiefly limited to the right side, but in the middle of the pons it involves both sides. Careful examination failed to disclose the reason for the hemorrhage.

Deiter's Nucleus Syndrome.

Lannois and Chèze[1] report a case showing the syndrome observed in disease of Deiters' nucleus, consequent upon a hemorrhage localized in the pons. This syndrome has been called the syndrome of Deiters' nucleus, and also Bonnier's syndrome, and is dependent upon a lesion of the nuclei of the pons, particularly of the external nucleus of the vestibular nerve (Deiters' and von Bechterew's nuclei). The essential symptoms are: (1) More or less intense vertigo; (2) inability to walk without sinking down on account of the giving way of the legs as in tabes and in labyrinthine disease, due to the connections between Deiter's nucleus and the cerebellum; (3) reflex oculomotor disturbances, such as paresis or paralysis of the sixth nerves, conjugate deviation, mystagmus, etc.; (4) nausea and vasomotor symptoms commonly found in pontine lesions; (5) transitory auditory disturbances ascribed by Bonnier to an associated lesion of certain fibers of the auditory root; (6) painful manifestations in the territory of the fifth nerve, especially in the temples, due to connections between Deiter's nucleus and the collaterals of the trigeminal sensory root. The essential symptom is the vertigo, due to a lesion of the nuclei of Deiters and Bechterew, the other symptoms being merely "neighborhood" symptoms, some attributable to lesions of the nerves moving the eyeball, especially the sixth cranial nerve, and some attributable to lesions of the pontine nuclei. Whatever the pathology, hemorrhage, tubercle, tumor, or gumma, the symptoms are fundamentally the same, though the "neighborhood" symptoms are very variable. To this syndrome belong the cases of Babinski and Frageotte, where in addition to the vertigo there was a hemiplegia, hemianesthesia, hemiasynergy, latero-pulsion, and unilateral myosis, all the latter symptoms being due to the involvement of one-half the pons.

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(1) Lyon Méd., Nov. 1, 1908.

The case now reported by Lannois and Chèze occurred in a man 65 years of age, giving a history of syphilis in early years and also of a left-sided pleurisy seven years before. While straining at stool he suddenly felt what seemed like a gunshot in his head. At the same time it seemed as though his eyes turned, and as though his mouth were pulled to the right. He became very dizzy but was able to reach his bed. He neither fell down nor lost consciousness. Severe headache followed with diplopia and persistent vertigo. The vertigo disappeared when he lay down, but reappeared as soon as he sat up, and reached the maximum when he stood. Examination showed the left corner of the mouth to be higher than the right, the tongue deviated to the right, the left external rectus muscle was paretic, there was diplopia, the left image being higher than the right. There was no paralysis or disturbances of either the tendon or cutaneous reflexes. If the patient was placed on his feet he could not stand up. Three months later he died. *Post mortem* there was found a hemorrhage at the external angle of the fourth ventricle, about one millimeter from the midline and one millimeter below the floor, in the region corresponding to the *eminentia teres.* The blood had been absorbed and had left a little serum-filled cyst the size of a wheat-grain, except that other traces of blood were found as a little yellowish line stretching back two or three millimeters behind the main lesion.

Such a location, between Deiters' nucleus and the nuclei of the seventh and sixth cranial nerves, explains all the symptoms. Very interesting is the fact that at the very first the right internal rectus was also paretic, this being confirmatory of the opinion advanced by Duval and Laborde of a common center for synergic movements of the eyeball.

DISEASES OF THE SPINAL CORD.

MENINGITIS SEROSA CIRCUMSCRIPTA.

A number of cases of meningitis serosa circumscripta have been reported during the past year, doubtless due to stimulation of investigation of this disorder by the writings of Oppenheim, and by the further fact that operation affords opportunity for complete cure. C. de Montet[1] reports a case, presenting symptoms of tumor of the cord, just as do all cases thus far reported. The patient began to complain of very intense pain in the back, especially upon the right side, in December, 1907. A few days later she felt numbness in the legs, the gait became limping and weak. By New Year's Day she could not walk at all, and dribbling from the bladder annoyed her. When examined in February she showed double ankle-clonus, incontinence of urine and feces, an almost total paraplegia, increased tonus of all the leg-muscles, increased tendon-reflexes, and both Babinski and Oppenheim phenomena. The infraumbilical abdominal reflex was absent on both sides. Subjectively there was a girdling pain and paresthesiæ in the legs. Objectively sensation showed changes below the epigastric angle. The vertebræ were of normal configuration, but the fourth and fifth dorsal vertebræ were sensitive to pressure. A diagnosis was made of compression of the cord, with possible involvement, at the fifth and sixth dorsal segments. An extramedullary, and probably a meningitic process appeared to be at the bottom of the trouble, by reason of the initial symptoms which were clearly meningitic. The diagnosis of extramedullary tumor seemed incompatible with the acute onset. Syphilis

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(1) Correspondenz-Blatt f. Schw. Aerzte, Nov. 1, 1908.

was denied, and there was nothing to suggest its presence.

The evil case in which the patient found herself led to an exploratory laminectomy. The dural sac was found to bulge, and it was opened. At once a very considerable quantity of cerebrospinal fluid spurted out. The cord within was found thinned and compressed. The vertebræ were intact. After being irrigated with normal salt solution the wound was carefully closed. The further course proved the case to be what it seemed at the operation, meningitis serosa circumscripta. By May, 1908, the patient was able to do washing, and all of her subjective and objective symptoms had disappeared.

The author calls attention to one point which he thinks may be of diagnostic value, namely, the variation from day to day in the sensory disturbances, which he believes to be caused by varying degrees of pressure.

A second case of meningitis serosa circumscripta is reported at length by Kurt Mendel and S. Adler[1]. The patient, a woman 36 years of age, began to complain of pains in the back and chest in July, 1907. By September there had developed pain over the spinous processes of the first and second dorsal vertebræ, Romberg's symptom, slight paresis of the right leg, and impaired sensation below the mammæ, these symptoms suggesting a tumor of the spinal cord. A careful examination made November 14, 1907, disclosed subjectively pain in the back extending to the right shoulder, and forward to the breast and region of the stomach; inability to walk or stand; a feeling of numbness in the legs; involuntary twitchings of the muscles of the leg, especially during the night; inability thoroughly to evacuate bladder and rectum. Objectively there were found visible twitchings in the muscles of the legs, especially the right; paresis of both legs, especially the right; the left patellar reflex exaggerated, the right, lively; Babinski's toe-phenomenon on the right side; Oppenheim's sign present on both sides; and sensory disturbances as shown in Figures 13, 14, 15.

A diagnosis, based upon spastic paraparesis of the lower extremities, the positive Babinski and Oppenheim phenomena, the characteristic sensory disturbances, bladder

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(1) Berl. klin. Woch., Aug. 31, 1908.

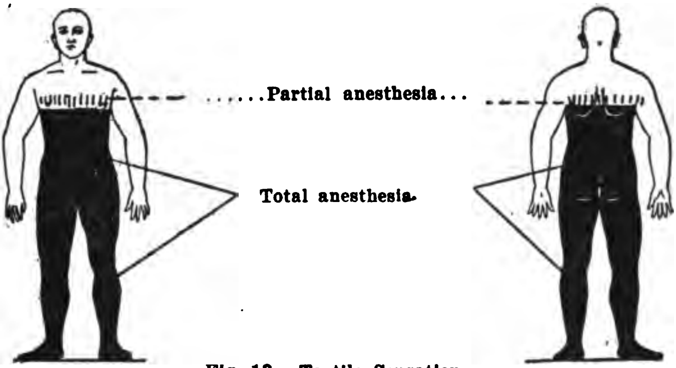

Fig. 13.—Tactile Sensation.

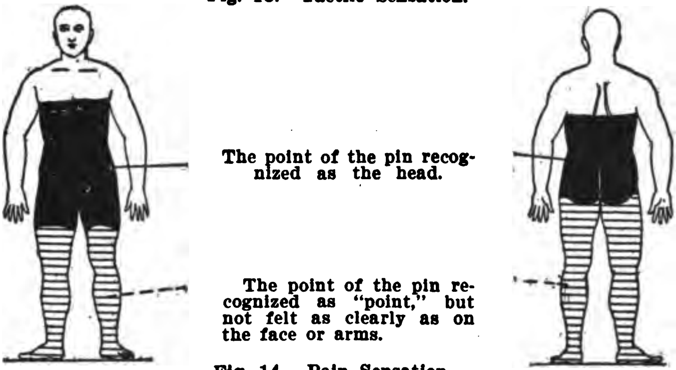
The point of the pin recognized as the head.
The point of the pin recognized as "point," but not felt as clearly as on the face or arms.

Fig. 14.—Pain Sensation.

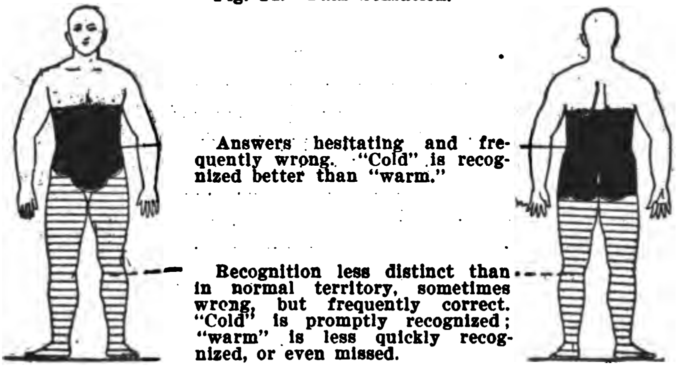
Answers hesitating and frequently wrong. "Cold" is recognized better than "warm."
Recognition less distinct than in normal territory, sometimes wrong, but frequently correct. "Cold" is promptly recognized; "warm" is less quickly recognized, or even missed.

Fig. 15.—Thermal Sensation.

and urinary symptoms, pressure upon the spinous processes of the second, third and fourth dorsal vertebræ, was made of a process causing compression from without upon the cord at the level of the second and third dorsal vertebræ. The sensory disturbances and the pains indicated this diagnosis. Since the symptoms predominated upon the right side, the maximum pressure was supposed to be there exerted. The process was thought to be an extramedullary tumor, but meningitis serosa circumscripta was thought of.

Operation was determined upon and performed in February, 1908, by S. Adler. On opening the spinal canal the dura welled up in the wound, but there was no pulsation. Careful palpation disclosed fluctuation but no tumor. When the dura was opened a small amount of cerebrospinal fluid flowed out, disclosing a violet-bluish transparent arachnoid bulging into the dural opening, but without pulsation. It was opened and there gushed out about one and one-half teaspoonfuls of cerebrospinal fluid, which was lost in the sponges. Immediately thereafter the cord began to pulsate. The subsequent history of the wound is inconsequential, drainage being carried on for twenty-four hours, and the wound closing by first intention in ten days.

An examination made July 4, 1908, showed only a slight weakness in the right leg, but no twitchings, a few pains in the back, no Babinski phenomenon, and a very great restoration of the lost sensations.

A very excellent resumé of the literature of this disease is given by Adler. After reviewing the original communications of Oppenheim and Krause, who in 22 laminectomies found meningitis serosa circumscripta 6 times, he calls attention to cases previously reported, Schlesinger's in 1898 as one of hydrops meningeus, that of Schwarz in 1897 reported in Vienna, Spiller's case reported in 1903 as one of intradural spinal cyst, and others. For those wishing to look into the literature carefully Adler's portion of the paper is especially recommended.

L. Bruns[1] having noticed the Mendel and Adler case, refers to it and to his own case mentioned in his book, "The Tumors of the Nervous System, Berlin, 1908," p.

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(1) Berl. klin. Woch., Sept. 28, 1908.

374. Adler believes most cases to arise by extension of an adjacent infectious process into the spinal meninges, as from caries of a vertebra, for example. But in four cases, Adler's own, Spiller's, a case of Krause's and Bruns' own case, no such associated infectious process was evident. These cases were apparently idiopathic. Bruns describes his case again. The patient was a schoolboy of 16 years of age, his first symptoms appearing in February, 1906, in the upper cervical region. Operation was performed by Boegel September 15, 1906. On opening the dura cerebrospinal fluid under pressure spurted out, but there was no tumor. The cord began to pulsate immediately after the escape of the fluid. Restoration to health followed, and now after a lapse of two years the boy remains well, with the exception of the Babinski phenomenon on the right side, a right ankle-clonus, and possibly a slight hypalgesia in the left leg. In view of the success in this case and others Bruns believes that cases of apparent tumors of the spinal cord ought to be operated upon, since there is a chance that such cases are really examples of meningitis serosa spinalis circumscripta.

A short paper upon the subject by W. G. Spiller[1] places the subject before American readers. Only one case is on record in this country, he says, it being reported by Spiller, Musser and Martin in 1903. Almost nothing is known as to the origin of apparently idiopathic collections of fluid within the pia-arachnoid. The symptoms are those of circumscribed compression of the spinal cord, and the condition cannot be distinguished clinically from tumor of the cord. The operation is easy and the cure apparently permanent in typical cases. The first case reported anywhere with operation was the case by Spiller, Musser, and Martin. In that case a delicate pial cyst was found and evacuated by D. E. Martin. The operation was on June 30, 1902. "The patient has been reëxamined by Dr. Musser and me within the past few weeks, and she has now no symptoms of her former trouble except slight pain in one buttock when she presses over the scar made by the operation. She has gained much in weight and is in excellent health. This is not only the first case with opera-

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(1) Amer. Jour. Med. Sciences, January, 1909.

tion, but is the only one on record in which recovery has persisted after operation so long as six years and five months."

A case of meningitis serosa circumscripta was unwittingly reported by M. A. Bliss[1] under the title: "Cysts Within the Spinal Canal." In August, 1908, the patient had "grip" (?), without cough or cold in her head, but she ached all over, most intolerably in her hips and back. About the same time severe cramping occurred in her legs. By the last of August she became extremely awkward in her gait. Loss of pain and temperature sense with fair preservation of tactile sense in her legs was also noted. About October 1 she became entirely paraplegic. The muscular contractures and startings were most severe.

Operation. By Dr. Harvey Mudd, November 30, 1908, 9 a. m. The spines and part of the laminæ of the seventh cervical, the first and second dorsal vertebræ, were removed. The dura bulged irregularly at the upper and lower ends of the part exposed, seeming to indicate a constriction between. The dura was a darker white at the bulging points. The dura was slit centrally from below upward. A sharp spurt of clear fluid, which could not be distinguished from the cerebrospinal fluid, occurred. It seemed that the lower end of a sack had been opened, for the upper end ballooned up with cerebrospinal fluid, as it surged upward in response to the cardiac movements. When the dura was grasped by the forceps and gently lifted, small bands of tissue were seen stretching between the dura and the pia. Very slight force with a dural probe sufficed to break these up, after which the ballooning process ceased. The dura was stitched. The wound was closed with a small catgut drain. Surgical recovery was prompt and uninterrupted.

By January 14, 1909, the patient could support her weight by placing her hand on a chair to steady herself. She could extend the legs voluntarily, but full extension caused discomfort. Her general health was perfect.

In the Interstate Medical Journal, May, 1909, M. A. Bliss again mentions his case, but under the caption "Circumscribed Serous Spinal Meningitis," his attention hav-

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(1) Jour. Amer. Med. Assoc., March 13, 1909.

ing been called to the true nature of his case by a recent paper of V. Horsley.[1]

TUMOR OF THE SPINAL CORD.

A tumor of the spinal cord presenting the symptomatology and course of myelitis is reported by Prof. C. A. Ewald and R. Winkler.[2] The patient, a motorman, 27 years of age, married a year, without history of alcoholism, syphilis or exposure to inclement weather, began to suffer with drawing pains between the shoulder-blades, these gradually increasing in severity and encircling the chest like a girdle. Subsequently there was frequent vomiting and gastric disturbances of transitory duration. Finally the pains appeared in the back, and the legs began to feel numb. On September 15, 1908, *suddenly* he could not move his legs very well, or void his urine. The next day by the help of his wife he was able to walk a little. When examined on September 17 by Ewald he showed no pupillary disturbance, nystagmus, or ocular palsy. There was sensitiveness in the epigastrium, over the bladder, and in both groins. The arms and hands were moved freely, but the legs showed a flaccid paralysis. Both knee-jerks were increased, but there was no ankle-clonus, patellar-clonus, or Babinski toe-phenomenon. The abdominal reflexes were absent, whereas the plantar reflexes were increased. No pain was caused by pressure on the spinous processes of the vertebræ. Sensation was intact down to the nipples. Below them the sensation for cold was completely, and for heat partially abolished. Tactile sensations were imperfect, and pain sensations in some parts absent. No zone of hyperesthesia was found. The next day the urine was passed involuntarily; and on September 19 the pulse rose to 120, the temperature to 39.1° C. (102.4° F.). A pneumonia developed in the right middle and lower lobes and the patient died four days later.

The post mortem September 25 showed a cylindrical tumor 4.5 by 5 cm. at the level of the seventh cervical vertebra, adherent neither to bone nor dura. This had compressed the cord very markedly, and on cross-section

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(1) British Med. Jour., Feb. 27, 1909.
(2) Berliner klin. Woch., March 22, 1909.

the latter showed numerous punctate and linear hemorrhages. Above and below the tumor the cord was normal.

In life the diagnosis was necessarily transverse myelitis, probably upon an infectious basis, both by reason of the sudden onset of the paraplegia (the day before he had driven his car as usual), and an enlarged spleen, found on physical examination and at the post mortem. Ordinarily when tumor causes cord symptoms by compression they are of very gradual development, stretching over days or weeks, with remissions and intermissions. The diagnosis of cord embolism in the case seemed to be supported by nothing. As for tumor, it could hardly have been supposed to be present. Yet a similar case has been reported by Nonne in a woman 40 years of age, who suddenly without special cause was seized with violent pain in the back, girdling in type, radiating into the thighs, associated with mild fever. Three weeks later while in the kitchen, after a few not very severe premonitory pains in the back she was suddenly paralyzed. This case was symptomatically one of transverse myelitis, but post mortem examination disclosed a sarcoma of the dura compressing the cord. Cases of a similar nature occur in cerebral tumors, a latent course being suddenly terminated by signs of hemorrhage or encephalomalacia in the form of an ordinary paralytic stroke.

SYPHILIS OF THE SPINAL CORD.

Acute syphilitic disease of the spinal cord, especially the so-called syphilitic spinal paralysis, is discussed by J. Collins and C. G. Taylor.(1) In 1892 Erb attempted to differentiate a form of disease of the spinal cord occurring exclusively in individuals who had previously had syphilis, resembling spastic spinal paralysis, but differing from it by the addition of some disturbance of bladder function and slight subjective and objective sensory disturbances.(2) Much has been written both in corroboration and in refutation of Erb's contention in this country and abroad.

There have been nine or ten cases of typical syphilitic spinal paralysis that have come to autopsy, showing a combined system disease consisting of a primary gray degeneration in various fiber systems of the spinal cord, chiefly in the posterior half of the lateral tracts, the direct cerebellar tracts, the tracts of Gowers, and somewhat also in the posterior tracts, the columns of Goll and Burdach. Nonne, in fact, has demonstrated most convincingly that although syphilitic spinal paralysis is clinically a well-defined entity, it has by no means an unvaryingly constant morbid anatomy.

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(1) Amer. Jour. Med. Sciences, February, 1909.

(2) See Practical Medicine Series, 1903, Vol. X, p. 177, for Erb's last paper on this subject.

The case reported herewith by Collins and Taylor is thus briefly summarized by the authors: "A youth aged 23 years, addicted to the intemperate use of spirits, became infected with syphilis September, 1907. Cutaneous and mucous membrane manifestations developed promptly. December, 1907, he had aphonia, due to syphilitic laryngitis. Six months after infection he developed weakness, stiffness and paresthesia of the lower extremities, and slight difficulty in urinating. The weakness of the legs intensified rapidly up to moderate paraplegia. This was promptly followed by retention of urine and later by incontinence of urine and feces, and impotency. After a very brief time he recovered sufficiently to leave the hospital, the symptoms at this time being those characteristic of syphilitic spinal paralysis. Cystitis and surgical kidney developed rapidly and he died eight months after infection, during which time he had been given mercury and iodid of potassium."

At the post mortem examination there were found "areas of softening, principally in the posterior half of the cord on transverse section extending from the first to the ninth dorsal segments, associated with changes in the blood-vessels, thickening and round cell infiltration of the vessel walls, circumvascular exudation, vascular thrombosis and hemorrhages; syphilitic myelitis; sclerotic patches due in part to growth of glia cells, principally toward the periphery of the cord, especially of the posterior and posterior lateral tracts; secondary degeneration of the posterior columns, especially in the column of Goll and also of the direct cerebellar tract and Gowers' tract in the upper thoracic and cervical regions; slight, but distinct, productive meningitis, patch-like distribution in many thoracic segments. The cells of Clarke's column were much degenerated; those of the anterior horn suffering to a much less extent, possibly the effects of sepsis.

"*Comment.* We do not maintain that this is an example of syphilitic spinal paralysis. It does not conform to that disease either in its onset or in its early symptomatic display. A slow and insidious onset is considered quite characteristic of the disorder which Erb described. In the case that we report the onset was abrupt and paraplegia developed within a few days; but the point to which we particularly wish to draw attention is that this paraplegia soon disappeared and the patient left the hospital. When he left the hospital he was clinically an example of syphilitic spinal paralysis. Had he been examined for the first time on his return to the hospital and without the history of previous paraplegia, we should have had no hesitation in making the diagnosis of syphilitic spinal paralysis. Moreover, had his course of improvement been unbroken by the septic condition which overtook him, there can be no doubt that he would have eventuated in a typical example of this disease. Therefore we venture to believe that the pathological findings of this case are of great importance as indicating the anatomical foundation of some cases of syphilitic spinal paralysis."

A Syphilitic Form of Multiple Sclerosis is reported by W. G. Spiller and A. H. Woods.[1] The patient, a woman 52 years of age, entered the hospital because of pain and weakness in the lower limbs. This she had noticed first five weeks previously, and five days after its appearance she began to have severe pain below the knees. Later she lost control of the bladder and rectum. The pupils were equal and regular, the iris reacted to light and accommodation, the grip of each hand was somewhat diminished, and the biceps and triceps reflexes were exaggerated. Movements of the legs which were edematous caused much pain, and they were kept flexed at the knees so that the knee-jerks could not be obtained. There was a suggestion of ankle-clonus on the left side. Atrophy of the right interossei was found subsequently. Sensation

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(1) Interstate Medical Journal, February, 1909.

seemed to be normal in all forms. The symptom-complex was therefore one of spastic paraplegia with contracture, and the pain was produced by the passive movement of the limbs, because of the contractures. The typical symptoms of multiple sclerosis were absent, such as scanning speech, nystagmus, and intention-tremor.

After her death there were found "numerous areas of sclerosis throughout the cervical and upper thoracic regions of the cord. Some of these resembled very closely the degenerated areas found in multiple sclerosis.

"The conclusions we have reached from our investigations of the case reported in this paper and other cases are:

"(1) Syphilis may in rare instances produce a symptomatology indistinguishable from that of typical multiple sclerosis, and this without the formation of sclerotic plaques, but by the ordinary lesions of syphilis, viz., arteritis and meningitis.

"(2) Syphilis may produce sclerotic plaques in the spinal cord resembling those of multiple sclerosis, without producing the typical symptoms of this disease. These plaques have not the sharp definition seen in most cases of typical multiple sclerosis, and yet multiple sclerosis may exist without plaques sharply defined from the normal tissue. The syphilitic form of multiple sclerosis presents round cell infiltration of the pia and thickening of the blood vessels."

HEMATOMYELIA.

Gauthier reports from the service of Prof. A. Pic[1] a case of traumatic hematomyelia followed by the development of probable syringomyelia. The patient, a man of 32 years of age, while carrying a weight of about 160 lbs., fell from a height of a little under five feet, and hurt his back in the region of the kidneys, and his right hip. He did not lose consciousness, but he could not get up or move his legs, and he passed the night where he was out-of-doors, stretched out on his right side, his leg in contact with the damp earth. The next morning he was found, taken home, and put to bed, where he lay for a month, the left leg being very painful, and the right, on the other hand, so insensible that he could feel nothing. Bladder and rectal disturbances lasted about a week. In four weeks the patient began to get about with crutches, then with canes, and ultimately without any means of support. Everything in the examination indicated a typical case of hematomyelia: the sudden paraplegia following a fall on the back, sensory disturbances of the Brown-Séquard type (anesthesia on one side and hyperesthesia on the other), the sphincteric disturbances on the part of bladder and rectum, and the gradual regression of all the symptoms except for certain spastic-paretic conditions of the right leg. The three phases of a hematomyelia were well illustrated: (1) the sudden and extensive onset of symptoms (2) the phase of regression, and (3) the final phase of fixation.

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(1) Lyon Méd., Oct. 11, 1908.

Such was the condition of the patient when seen seven months after the accident, except for two other very important findings: (1) There was ankle-clonus with *loss* of knee-jerk, though the Achilles-jerk was present; (2) there was inequality of the pupils and nystagmus, the latter being difficult of explanation. At any rate these findings suggested to the author and Prof. Pic the probable evolution of syringomyelia. In this disease pupillary inequality is not rare, and nystagmus according to Schlesinger occurs in 10% of all cases. It is true that much is lacking of the essential signs of syringomyelia, yet the author believes it to be developing.

SYRINGOMYELIA.

Monoplegic Syringomyelia is discussed by A. Wemmer[1] and three cases are reported, one of which was purely sensory. This case was first seen in 1905, and occurred in a shoemaker 38 years of age, beginning with paresthesia in the left thumb. Soon there followed thickening of the skin of the thumb, and torpid, painless and very slowly healing ulcers appeared. Later the whole left hand showed a cyanotic swelling. Subsequently he felt pain in the left arm, but never was there a trace of paresis or muscular atrophy. With the exception of mild horizontal nystagmus, there was nothing in the cranial nerves. The spinal column was normal, and the x-ray showed no syringomyelic modifications. The right arm was wholly normal, both as to sensation and motion. The sensory changes in the left arm are illustrated in the following figures, 16, 17 and 18. It will be noted that the sensory disturbances are suggestive of the radicular type.

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(1) Berliner klin. Woch., Aug. 26, 1909.

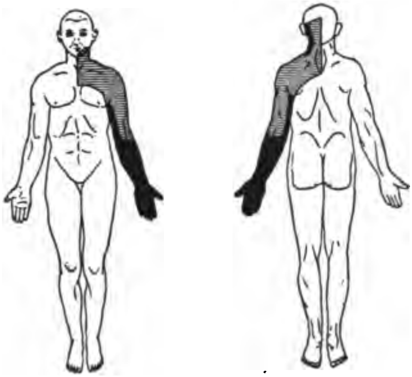

Fig. 16.—Thermanesthesia.

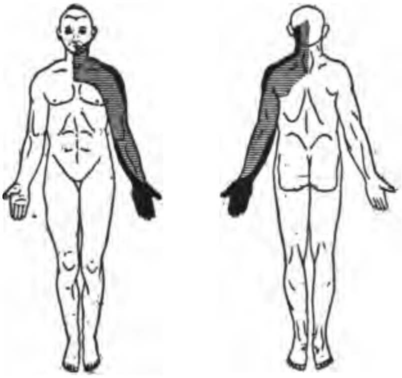

Fig. 17.—Analgesia.

Two other cases of syringomyelic monoplegia, with both sensory and motor symptoms, one in the right arm, and the other in the left leg, are reported. Cases of unilateral syringomyelia are rather rare, and monoplegic cases still more so. Of late Petrén has reported a case of purely spastic type with symptoms of syringomyelia almost exclusively unilateral,[1] in which report is a very complete review of all similar cases thus far reported.

Laryngeal symptoms rarely appear in syringomyelia, only 26 cases being reported in medical literature, and a 27th case being now added by E. Baumgarten.[2] In syringomyelia the first laryngeal symptom to be observed is hoarseness, and in the case reported it was this which made the patient consult a physician. He was 39 years of age, was hoarse, and his voice had a peculiar eunuch-like sound. On examination both vocal cords were found fixed nearly in the median line, and in deepest inspiration showed no outward motion. The right side of the tongue was considerably thinner than the left. There was no atrophy of the lips. Sensory disturbances (thermanesthesia) were found in the right hand and forearm, and the pain-sense was reduced over the whole right upper extremity. There was, moreover, atrophy of the interossei and thenar muscles. The right palatal arch was paretic. Curiously enough from one ear to the other there ran a band of thermanesthesia, around the lower jaw. Thus not only the cord but the medulla as well was involved. Schlesinger first collated

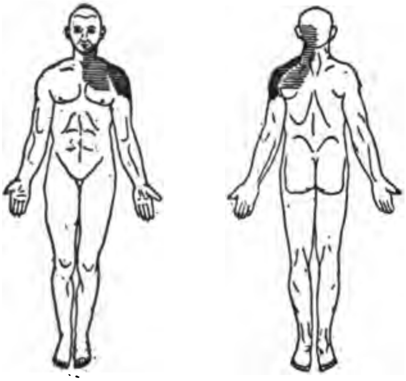

Fig. 18.—Hypesthesia.

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(1) Deutsche Zeitsch. f. Nervenheilkunde, 1909, Vol. 63, p. 400.
(2) Berliner klin. Woch., Aug. 28, 1909.

these cases, Baurowicz added to them to the number of 26, and Baumgarten at last adds another to the few, very briefly reporting all 27 cases in so far as they relate to the laryngeal lesion. In 3 of the 27 cases the cords were not paralyzed, but the laryngeal reflexes were either entirely lost or greatly reduced, and they were more or less anesthetic. Atrophy of the tongue occurred in 9 of the 27 cases, and in Baumgarten's case.

MULTIPLE SCLEROSIS.

Etiology. Multiple sclerosis developing after psychic shock is reported by G. W. Wallbaum.[1] Von Leyden, three decades ago, declared it to be his opinion that psychic trauma might cause paralysis agitans, myelitis, and paralysis. Fright has long since been thought to cause multiple sclerosis, and Wallbaum reports three cases from his own practice where fright was the apparent cause of onset. He attempts to show that not one of his three cases had a symptom of multiple sclerosis before the psychic shock, though one of them possessed neurasthenic symptoms. After discussing the subject at length, the author concludes: "It is certainly true that multiple sclerosis may develop after psychic trauma. The only necessary basis for such a possibility is a predisposed nervous system. In all probability multiple sclerosis after shock develops upon a vascular basis, and myelitic processes may be interpreted as prodomata of the disease when it so originates."

LOCOMOTOR ATAXIA OR TABES DORSALIS.

Etiology. Concerning the etiology of tabes F. W. Mott[2] expresses himself as follows: "The Wassermann method of diagnosis has come to strengthen and confirm the belief of many neurologists like myself: 'No syphilis, no tabes.' This was previously based solely upon statistics and observations relating to the etiology of the disease. Moreover, the etiology and the serum diagnosis are reciprocally supporting not only of the parasyphilitic theory, but also of the view that there is one morbid entity which

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(1) Deutsche med. Woch., Dec. 10, 1908.
(2) British Med. Jour., Feb. 20 and 27, 1909.

may be described as tabes—and but one cause—syphilis, acquired or congenital; and that there are a number of contributing factors, any one of which by itself, or even in combination with others—for example, sexual excess, mental stress, heredity, injury, etc.—is not capable of producing the disease.

"Although syphilis is the essential cause, yet, as Fournier showed, tabes and general paralysis are not syphilitic, but an outcome of syphilis, and the riddle is still unsolved why only about 3 to 5% of the persons infected with syphilis should subsequently suffer with one of these degenerations of the nervous system termed parasyphilitic."

Symptomatology. *Gastric crises* in tabes with vomiting of blood are reported by J. Kollarits[1] with post mortem findings in a case. The crises occurred during the last weeks of life, and blood was vomited, and once there was a bloody stool. The bleeding was so profuse that ulcers or carcinoma was suspected, but at the autopsy only the typical changes of tabes were found. Hence there is such a thing as a parenchymatous hemorrhage as a rare accompaniment of a gastric crisis. Similar cases of bleeding, but not in tabetics, have been described by Reichard.[2]

Tolot and Froment[3] have made a study of the pathologic condition of the stomach in those cases of tabes having had gastric crises, which have come to post mortem examination. Gastritis is not rare in subjects with tabes and gastric crises, due often to alcohol and sometimes to the drugs which have been taken, particularly potassic iodid; and each tabetic with gastric crises has to endure them with the mucous membrane which he possesses, as has been aptly observed. The authors found only 17 cases of tabes in which both gastric crises and post mortem findings were recorded; and of these 17 there were only 9 where the pathologic record as to the stomach was noted with any precision. These 9 cases divide into three groups. In group I are three cases, those of Graves published long ago under the caption "A Case of Paraplegia," that of Rendu reported in 1890, and an unreported case of Devic's recorded in this article for the first time. In all of these

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(1) Neurolog. Centralblatt, Jan. 2, 1909.
(2) Deutsche. med. Woch., 1900, p. 827.
(3) Lyon Méd., Feb. 21, 1909.

the macroscopic examination of the stomach was complete and absolutely negative.

Group II consists of three cases in which slight alterations were found, the exact significance of which it is hard to estimate. In Tripier's case, reported in 1882, the stomach was very much dilated. In Pierret's case, quoted by Putnam in his Paris thesis in 1882, the stomach was somewhat slate-colored, due to the deposit of coloring matter in the submucous connective tissue. [This is suggestive of the old silver nitrate medication used in tabes.—Ed.] The third case, reported by Déjerine in 1883, showed what he called a "gastromalacia of the fundus." These three observations, dating far back, are hard to classify.

Group III comprises three cases. The first, Jean's case, reported in 1876, in the Progrès Médical, was characterized by constricting pains of great severity at the level of the epigastrium, sometimes accompanied with nausea and vomiting. Abundant hematemesis took place, accompanied by laryngeal and pharyngeal spasms, just before death. At the post mortem examination the stomach was found very much contracted from the median line to the pylorus, being no broader than the duodenum; its walls were very thick, measuring 8 mm.; the mucous membrane at the anterior border of the great curvature showed superficial ulcerations. The second, Crouzon's case, reported in 1906, showed for fifteen years classic gastric crises, and blood was frequently vomited. The vomitus was a brownish liquid, and contained coffee-ground particles. The post-mortem examination showed the stomach to be very much shrivelled, reduced practically to a straight tube, with walls extremely thickened, but with no recent or obsolete ulceration of the mucous membrane, even though there was vomiting of blood just ten days before death. Microscopic examination showed infiltration with leucocytes and a subacute gastritis. The third case, that of Roque and Chalier, follows.

A case of cancer of the stomach with gastric crises in a tabetic is reported by Roque and Chalier.[1] Properly speaking the pain was not really in the form of typical gastric

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(1) Lyon Méd., Dec. 20, 1908.

crises, but there were acute paroxysmal pains, which for a time obscured the diagnosis of carcinoma ventriculi. At first the seizures were far apart, but subsequently they came nearer together, until they were of daily occurrence, usually starting in about 8 P. M. and lasting until 3 or 4 A. M. In this case it was not until a nodule was found near the region of the pylorus that the diagnosis of carcinoma was made, this being about three months before the patient's death. Post mortem examination showed carcinoma of the stomach with metastases in the liver, and tabes dorsalis.

Nasal Crises. The nasal crises of tabes form the subject of an elaborate study and paper by Klippel and Lhermitte.[1] Disturbances of the sense of smell and taste were reported in tabes dorsalis as far back as the time of Charcot and Vulpian, but not until recent years have they been sufficiently studied. The nasal crises may be divided into three groups: (1) sensory, (2) motor, and (3) vasomotor or secretory. Though these groupings are to quite an extent schematic, a given case showing a crisis belonging to two groups at once, still they are very convenient for purposes of orderly description.

(1) Sensory crises in tabes consist in a diminution or suppression of the sense of smell in one or both nostrils. Associated with the total or partial anosmia there are apt to be sensory disturbances affecting the face and nasal mucous membrane. In general the crisis comes on without appreciable cause, with a pricking sensation at the root of the nose, a tickling sensation in the nasal mucous membrane, and then the fundamental phenomenon of the nasal crisis, namely, the perception of an odor very strong or very disagreeable, often compared to the odor of bad fish or rotten eggs. In some cases there is no sensory aura, a case in point being cited. In a few rare cases the crisis may come on in sleep, disappearing upon awaking. So burdensome was this in the case of one patient reported that he slept as little as possible to avoid the intolerable sensation. The diagnosis of this condition, given the signs of tabes, is easy. It must be distinguished from the olfactory aura of epilepsy and gross brain disease, and from the parosmias of certain mental diseases, especially paranoia.

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(1) La Semaine Méd., Feb. 17, 1909.

Often this particular form of nasal crisis ushers in a case of tabes dorsalis.

(2) The second form is the motor or spasmodic type of nasal crisis, which is seldom pure, being nearly always accompanied by sensory symptoms as well. Two cases were cited, one beginning with an aura of a pricking sensation at the root of the nose, followed in a few seconds by an outburst of sneezing lasting without interruption for three or four minutes. During the period of sneezing no particular odor was smelled, and there was no flow of nasal mucus, such as takes place in spasmodic rhinitis independent of tabes. These crises may occur early in tabes, or even very late. They may occur with a perfectly dry nasal mucous membrane.

(3) The third form is the vasomotor or secretory. This really occurs in two subvarieties, the spasmodic rhinorrheic type, and the pure rhinorrheic type. In the former there is a crisis of sneezing accompanied by an abundant flow of mucus. In the latter there is merely an abundant secretion of mucus without apparent cause, occurring intermittently. The diagnosis of these two forms is simple, nasal hydrorrhea in Bright's disease being excessively rare and wholly unlike that of tabes, and nasal hydrorrhea in the course of brain tumors, due probably to intracranial hypertension of cerebrospinal fluid, being easily eliminated.

When examined carefully the olfactory nerve filaments in these cases of nasal crises in tabes, show a partial degeneration of the fibers from the olfactory bulb to the hippocampal gyri and the corpus callosum. The trigeminal nerve probably plays an accessory part in the genesis of tabetic parosmias, and lesions have been found in the Gasserian ganglion in a case showing a diminution in the smelling capacity. The authors believe it likely that in all cases of nasal crises the trigeminal nerve is affected, and possibly also the sympathetic. They believe that no nasal crises occur if the olfactory apparatus and the trigeminal nerve are free from pathologic changes.

Treatment. The treatment of tabes dorsalis is discussed by A. M. Hamilton.[1] When the diagnosis of tabes

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(1) Jour. Am. Med. Assoc., Dec. 5, 1908.

is made a gloomy prognosis should not be given, for the mental point of view is of vast importance. Indeed, an apparent improvement attends the use of any new remedy, especially if novel or mystic, and solely by virtue of its mental effect. In only a very small proportion of cases is the use of mercury or potassic iodid of any avail, but they are occasionally of service in rapidly developing cases. "I have for years used, either in small doses by the mouth or by inunction by means of some readily absorbable agent, the bichlorid of mercury with encouraging success. By inunction it is superior to any of the oleates or better-known salves or emulsions."

The treatment of pain is often most difficult. Pyramidon is about the best of the coal-tar remedies, but none of them is of much use except in dangerous doses. If the pains are affected by variations of barometric pressure aspirin or one of the salicylates may be recommended. Morphin used under a physician's direction need not produce a habit. Prolonged warm baths are useful.

The ataxia may be kept in abeyance or overcome by systematic exercise, as suggested by Fraenkel's method. The idea is to train the individual so that he may be able better to coördinate. As a rule the persistent employment of regular exercise teaches the patient to train his power of balancing and the direct corrective use of vision and touch, as well as precision in consummating particular acts, do much good. The evil effects of fatigue must never be lost sight of, however.

In about 60% of cases the bladder is diseased, and attention should always be paid to it. A case is reported, showing extreme symptoms of tabes, in which great improvement ensued when the bladder was properly treated.

J. R. Jacoby[1] recounts the result of his recent visit to Heiden, where he saw Fraenkel's treatment of tabetics by the reëducational method. "In the education of the patients in the gymnasium, practically no apparatus is used. With the exception of a constructed staircase on which the patient is taught to go up and down stairs, the only aid used is a series of markings on the floor. The hall is about 15 meters long and on the floor are marked trans-

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(1) Med. Rec., May 8, 1909.

verse lines that can be distinctly seen, and the distances between these lines represent variously what Fraenkel terms ¼, ½, ¾, and full steps. On these limited spaces the patient is taught to walk his measured step, which is varied from time to time in length and rhythm. Variations of equilibrium are effected by placing the arms in different positions. Fraenkel decries the use of gymnastic apparatus, whether simple or complicated. He reiterates that the reëducational treatment is to *educate* muscles, not to strengthen them. The muscles generally, in tabetics, are sufficiently strong and well developed, in fact the muscle-play is always very much in excess of the actual force required to effect any desired movement. The use of weights and appliances, Fraenkel maintains, can seriously injure the patient, as it subjects him to a strain that not only exhausts the muscle fibers, but dulls the remaining sensibility inherent in the muscle itself. The theory that if a person must jump five feet, he should try to jump ten, does not hold good in the reëducational treatment. Nothing more should be required of the patient than is actually necessary to accomplish any particular movement. And it is for this reason that the exercises are so carefully graded."

Six cases seen at Heiden are then reported as to the treatment. The author concludes: "From my observations of these and the many other cases that I saw and cared for in Heiden, I have come to the conclusion that a majority of the tabetics can be materially helped by the reëducational treatment, that the greater the ataxia, the more benefit there is to be derived from this form of treatment, and that the prognosis is in a measure dependent upon the hypotonia of the muscles.

CAISSON MYELITIS.

Two cases of Caisson disease in divers are reported by Wassermeyer from Siemerling's Clinic at Kiel.[1] A torpedo had been lost and the two divers had been searching for it in 100 to 110 feet of water (4 to 5 atmospheres of pressure). One of the patients, after working three days, and after being under water four hours, came to the sur-

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(1) Berliner klin. Woch., Feb. 8, 1909.

face as usual, and on trying to urinate five minutes after he was up, could not do so. He at once began to feel dizzy and was obliged to sit down. In about ten minutes he was a little out of his mind. From the small of the back downward his body felt numb and prickly. He could not walk and was carried home. For five days he could not urinate, and had to be catheterized. He was also unable to move his bowels, high enemas becoming necessary. The pains in his back and legs became severe. The knee-jerks were increased, patellar clonus was not present, the Achilles-jerks were increased, on the right side there was a suggestion of ankle-clonus. His condition gradually improved from July 7, the date of the seizure, until May 20 of the following year, when he showed increased knee-jerks, patellar clonus, uncertain spastic-paretic gait, and Romberg's sign, he still being far from well.

His fellow-diver, taken the same day after two hours under water, was not so severely stricken. After he had been up about ten minutes, on July 7, 1907, he began to have cutting pains in the body and trembling in the legs. He could no longer stand. His legs then became stiff and numb, and somewhat cold, and though he could walk by holding on to things, still he was very weak and felt sick. By night he could not walk up a flight of stairs. He had retention of urine for nine days and was obstinately constipated. He showed increased knee-jerks, patellar clonus, ankle-clonus, and a doubtful Babinski toe-sign. There was no Romberg's sign, and he could walk very well. No spasms, atrophies or electrical reactions of degeneration were present. By the following May (1908) he had improved very little. He complained then of pain in the scrotum and buttocks, he had to urinate very slowly, and he could not defecate normally. If he bent over very long he had transitory vertigo. The reflexes, normal and pathologic, were unchanged.

Evidently in both cases there was a posterior and lateral cord involvement. The localization of the process at the onset was in the dorsal cord. According to the author, under the increased pressure considerable amounts of gases, especially nitrogen, are absorbed by the blood; and these gases upon the sudden release of the pressure are liberated, thus causing capillary emboli, especially in the terminal arteries of the spinal cord. No hemorrhages take place, but the expanding gases damage the nerve-tissues. Wassermeyer thinks that the great difference in temperature between sea-water at the depth of 100 feet and the air is a contributing factor. Chilling of the surface of the body will empty the superficial vessels and swell the deep-lying ones.

ACUTE ANTERIOR POLIOMYELITIS.

An epidemic of acute anterior poliomyelitis occurring in Salem, Va., and vicinity, is reported by R. M. Wiley and J. C. Darden,[1] a number of individual case histories being added. The epidemic occurred in the summer of 1908. The first case developed June 2, and the last August 10, and not a single case was reported from any other place in the state. There were 25 cases in Salem and the adjacent country, whereas in Roanoke, a city of 35,000 inhabitants, seven miles east of Salem, connected by a trolley and daily visited by scores of Salem people, not a case developed, during this period, though there was one case in September.

Clinical Case. A peculiar case of acute anterior poliomyelitis is reported by F. Carles and Desqueyroux.[2] The case is reported because of the involvement of certain muscles rarely affected, of certain deformities seldom produced, and because of the existence of progressive muscular atrophy of the Aran-Duchenne type. The patient, a boy of seven and a half years of age, two weeks after being vaccinated, and one week after going swimming, was suddenly seized with pain in the head and back. Chilly sensations and a high fever followed, together with a generalized paralysis involving all the muscles of the body,—arms, legs, trunk, neck, head. The patient could not urinate and had to be catheterized for four days. The muscles of the eyes, throat and larynx were not affected, and he could breathe normally. The paralysis lasted three months. From the beginning the fingers of the right hand were flexed in the palm; later the hand opened and muscular atrophy of the Aran-Duchenne type was observed. The

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(1) Jour. Am. Med. Assoc., Feb. 20, 1909.
(2) Jour. de Méd. de Bordeaux, Sept. 27, 1908.

same thing to a less degree occurred in the left hand. The intelligence was in every way intact.

There were some points about the case of an exceptional nature. First, the duration of the paralysis for three months was excessively long, most cases being completely paralyzed only a few days. Second, the sphincters were involved, there being paralytic retention of the urine. Third, many groups of muscles almost invariably spared were affected, notably the muscles of the left abdominal wall and of the left lumbar region. The left-sided paralysis of the abdominal wall, with an intact right wall, produced a pseudoventral hernia; for as the right wall contracted, it forced the bowels to the left, where the weakened wall permitted bulging to take place. Moreover, the paralysis of the left lumbar spinal muscles produced a skoliosis by the overaction against the tonic resistance of the right lumbar muscles. Thus the convexity was to the left. Lastly, the case is peculiar in that the hands showed the type of muscular atrophy known as Aran-Duchenne. These last three peculiarities, pseudoventral hernia, skoliosis, and progressive muscular atrophy of the Aran-Duchenne type have all been reported in infantile paralysis, but probably never combined in one case.

DISEASES OF THE PERIPHERAL NERVES.

Cranial Nerves.

Tumor of the Gasserian Ganglion. A report of two cases is made with post mortem findings by W. G. Spiller.[1] Tumor arising in the Gasserian ganglion is rare, but more common than is generally supposed. When it occurs, pain is intense. The first case of tumor in this region recorded in this country, and the second case on record in which operation was performed is that reported by Dercum, Keen, and Spiller in 1900. (J. A. M. A., April 28, 1900.) Since then Spiller has had in his care two more cases with post mortem findings, in one of which operation was performed; and it is these which he now reports. Without going into detail it is enough to say that the important

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(1) Amer. Jour. Med. Sciences, November, 1908.

features of the first case were as follows: "Paralysis of the right fifth nerve, with severe pain as the first symptom, then blindness of the right eye, later of the left eye, with optic neuritis, impaired mentality when the pain was severe, inequality of pupils, the right being the smaller, loss of reaction to light probably from the optic nerve disease, bilateral deafness, weakness of the soft palate in the right side, impairment of the sense of smell, loss of patellar and Achilles tendon reflexes, ataxic gait and station, weakness of the left third nerve, still greater weakness of the right third nerve, and paralysis of the right sixth nerve. The facial nerves were not affected. The possibility of syphilis was at first considered, but later tumor of the Gasserian ganglion was the diagnosis." By reason of the severe pain a decompression operation was done, but the patient died the next day.

Spiller himself removed the brain, and found the right temporal lobe softened, and tightly adherent to the floor of the middle cerebral fossa by reason of a tumor which seemed to grow from the dura, and was most developed at the seat of the right Gasserian ganglion, nothing of which was to be made out. The tumor covered the floor of the middle fossa, covering the foramen ovale, foramen rotundum, and the foramen lacerum medium, as well as the inner part of the petron in the posterior fossa mesial to the internal auditory meatus. The tumor was firm and measured about 4 cm. in each direction, and extended about 3 cm. into the brain. The right fifth nerve was enveloped in the tumor at its entrance into the pons, and the right third, fourth and sixth nerves were also caught in the tumor. It extended so far to the left as almost to catch the left third nerve.

The important features of the second case were: "The history of a second exposure to probable syphilis, the pain and weakness in the distribution of the right fifth nerve, impaired hearing on the right side, lessened sweat secretion in the right side of the head, inequality of the pupils, sluggish iridic reaction to light, with prompt reaction in accomodation, weakness of the right external rectus muscle, some retraction of the right eyeball, and narrowing of the right palpebral fissure." Examination of the nose showed a tumor in the nasopharynx, and this seemed to point to an intracranial tumor as the cause of the symptoms. This possibility was strengthened by the excision of an enlarged lymph gland of the neck, and the discovery of an endothelioma within it. Hence Spiller made a diagnosis of tumor inplicating the right Gasserian ganglion, probably endothelioma.

Operation promised little, because it was evident that the tumor was extensive, was both intracranial and extracranial, and its complete removal was impossible. Pain in the right fifth nerve distribution was so intense, however, that relief was imperative. Dr. Frazier accordingly removed a portion of the affected Gasserian ganglion. About seven weeks later the patient died, and the tumor was found to have been correctly localized.

In Spiller's case reported in 1900 the glands of the neck were found early enlarged, and some of these glands were found to contain endotheliomatous tissue. Such glandular enlargements furnish a valuable means of diagnosing intracranial neoplasm.

The diagnosis of a lesion of the Gasserian ganglion is usually easy, but it may be difficult to determine whether it is a syphilitic meningitis or a tumor. One is tempted, given a history of syphilis, to decide in favor of syphilitic infection, both by its frequency as a cause and his desire to make a diagnosis where there is some hope of relief by medication. Tumor of the Gasserian ganglion seems in most cases at least, to be irremovable. It apparently originates in the dura, and has the character of an endothelioma or sarcoma. It is usually of large size, extending as a flat growth, over the base of the middle fossa, possibly into the posterier fossa, possibly into the orbit.

FACIAL OR TRIGEMINAL NEURALGIA.

Diagnosis. The diagnosis of facial neuralgia is discussed ably by J. A. Sicard.[1] The term facial neuralgia should be limited, and the author proposes the following classification for all cases:

"1. Essential facial neuralgia.
2. Secondary facial neuralgia.
3. Facial neuralgism."

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(1) La Presse Méd., April 21, 1909.

This new word seems necessary to Sicard since it expresses the fact of the existence of a clinical syndrome often met with and quite characteristic.

Essential facial neuralgia is that form without a discoverable cause, but the cause of which doubtless lies in the deep or peripheral foramina through which the great nerve trunks pass. It is a very frequent form, comprising from 80 to 90 per cent. of all cases. Sicard believes it even now possible to ascribe essential facial neuralgia to compression of nerve trunks at certain foramina, by reason of some badly defined hyperplastic osteoperiostitic processes.

Secondary facial neuralgia always has a discoverable cause, which may be local (central or peripheral), or general. The latter are generally syphilitic, diabetic, or malarial.

Facial neuralgism is a term used for such prosopalgias as are due neither to essential nor to secondary facial neuralgia. The diagnosis of this syndrome is to be made only by exclusion. Sicard in schematic form outlines the essential characteristics of each of these forms of facial neuralgia.

Secondary Facial Neuralgia.	*Essential Facial Neuralgia.*
An apparent cause, especially of a *peripheral* origin, such as abscess, sinusitis, adenopathy, neoplasm, specific gumma, actinomycosis, etc.	No apparent cause. Local examination negative. Extraction of teeth an illusion.
A *central* origin; often simultaneous involvement of all three branches of the trifacial nerve, with possibility of reaction on the part of the motor masticatory branch, or its paralysis; associated diplopia, deafness.	At the beginning all three branches are never simultaneously involved. A delay of several years is often necessary for the extension of the pain to a neighboring branch. The involvement, even successively, of the three branches is very rare. Trismus, paralysis of the motor masticatory

	branches, diplopia, deafness, never occur.
Hypesthesia frequent, or even anesthesia in one or more of the cutaneous or mucous territories of supply by the trifacial nerve.	Hypesthesia and anesthesia never present. Hyperesthesia is frequent.
General causes; *diabetes*: presence of sugar in the urine and involvement of other cranial nerves;—*syphilis*: likewise participation of other cranial nerves and cerebrospinal lymphocytosis. Yields in general to antidiabetic or antisyphilitic treatment.	No sugar in the urine; never involvement of other cranial nerves; no cerebrospinal lymphocytosis. Essential facial neuralgia may exist among syphilitics and diabetics without yielding to appropriate treatment.
Pain often continuous with spontaneous paroxysms.	Pain not continuous, with a remission of a few hours at the beginning, with very short remissions at the period of confirmed neuralgia. Paroxysms extremely violent, often awakened by a movement in the face.
Follow the causal localization, possibility of talking, masticating, swallowing, without arousing too much pain.	In the crisis absolute impossibility to speak, masticate, or swallow.
As a general rule the hyperemic phenomena progress continuously, and the crises are not so strikingly paroxysmal.	Vasomotor hyperemia of half of the face by reason of congestive storms during the course of the crisis. Vasodilatation of the conjunctiva, lachrymation, salivary hypersecretion, likewise occurring like little squalls during

	the paroxysmal crisis of the main storm.
In neuralgia of central origin, there may be an associated headache (cerebral compression).	Never headache.
No localization of election on the part of the pain. Often diffuse pain.	Special localization of the pain: infraorbital foramen, supraorbital or mental. Fixation of the pain at one or more teeth. The patient points out clearly with his finger the course of the painful nerve ("the pain comes back to die in its hole just like the rabbit."—Brissaud).
No special deduction to be made from local tests, contact, or deep compression.	Contact with skin or mucous membrane causes more of a crisis than does firm deep compression.
Muscular twitchings, and local myoclonias are rare.	Tonic and clonic motor phenomena very frequent. Fibrillary muscular twitches with little spasm-like contractions. It is really spasm and not tic, as Brissaud has shown.
Duration limited by further progression or the cure of the malady.	Duration unlimited, 25, 30, 40 years.
Appears indifferently on the right and left side.	Much more frequent on the right side.

Facial Neuralgism.	*Essential Facial Neuralgia.*
Pains not well localized, diffuse. Often predominance in the temporal region.	Pains always well localized. Rarely predominance in this region.
Often bilateral.	Always strictly unilateral.

Frequent participation of pains in the neck (Arnold's nerve), and of the cervical plexus.	Rare possibility of participation of the suboccipital nerve, but no participation on the part of the cervical plexus.
No cutaneous hyperesthesia. The skin of the face is taken care of.	Cutaneous hyperesthesia is very frequent. Contact with the finger, the garments, a razor, is sufficient to provoke a crisis. Carelessness about the facial appearance.
Pains recur frequently at a fixed hour (habit pains of Brissaud).	Pains most often without relation to morning or evening hours.
Pains often calmed by mastication or the ingestion of food.	Pains made worse by the use of the buccal, labial. lingual and pharyngeal muscles, etc.
Facial expression preserved even at the time of the pain. No myoclonic jerkings nor muscular twitchings.	Impassive facial expression, the face rigid, during freedom from the pain. Often partial myoclonia during the crisis.
Psychasthenic state.	State of resignation."

In general, every facial neuralgia, not treated locally, accompanied by more or less extensive anesthesia in the trifacial territory, or associated with reactions on the part of other cranial nerves (diplopia, trismus, deafness, facial paralysis), is not an essential, but is a secondary neuralgia.

Treatment. Facial neuralgism, no more than migraine, should be treated by local injections. Sometimes such injections not only do not cure, but aggravate the pain. Secondary facial neuralgia is benefited by such injections only when the origin of the pain is peripheral. If the lesions are central (ganglionic or meningeal) the result is nil. Essential facial neuralgia is *always* cured by superficial or deep injections, provided the branches at fault are directly reached at their superficial or deep foramina, and provided that their nerve-elements are destroyed. The cure is ordinarily transitory, varying from 10 months to 2 years. The more thoroughly the trunks are injected, the longer the cure. The only indication of the thoroughness of the injection is afforded by the resultant anesthesia in the trifacial territory. The injection fluid should be 80 per cent. alcohol, or a solution of stovain or novococain of a strength of 1 centigram to 1 c. c. (gram). If alcohol fails *oil-injections* may be made. The formula is:

Oil of sweet almonds, 90 cubic centimeters.
Creosote.
Guaiacol, of each, 2 to 6 grams.
Iodoformed ether, 10 cubic centimeters.

The amount to be injected is not given.

The diagnosis of facial neuralgia is also discussed by F. Lévy,[1] stimulated by Sicard's paper. Lévy is inclined to divide all cases into two groups, little facial neuralgia, or neuralgia facialis minor, and neuralgia facialis major, or Trousseau's disease. He differentiates these two types thus:

Neuralgia Facialis Minor (Valleix's Disease.)	*Neuralgia Facialis Major (Trousseau's disease.)*
1. Pain continuous.	1. Pain not continuous.
2. Dartings of pain intermittent, always present, but relegated to a secondary rank.	2. Lightning-like dartings of pain, grouped in accesses, sometimes anticipative and simulating a continuous pain; brought back by the slightest effort, movement, talking, masticating, swallowing.
3. Painful spots on pressure, at the exits of the nerves, and persisting even when the paroxysms have eased.	3. No painful spots on pressure over the exits of the nerves (but sometimes alveolar bony points).
Especially localized in the supraorbital, inferior maxillary, more rarely in superior maxillary di-	In general localized in one branch, especially the superior maxillary, with consecutive invasion of

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(1) La Presse Méd., June 9, 1909.

visions of the trifacial nerve.	the first and third divisions of the nerve.
Very rarely, disturbances of objective sensibility (radiculo-ganglionic neuritis), hypesthesia, anesthesia. Exceptionally present in basilar neuralgias (Gasserian syndrome).	No disturbance of objective sensibility. Probable appearance at a certain period of the malady of spasms, tonic or clonic, at first voluntary, munching, grimaces, then involuntary (*tic douloureux*).
Involvement of the three branches, and other cranial nerves, (iii, vi, vii, etc.).	Manifestation of secretory and vasomotor disturbances.
Causes frequently peripheral, teeth, nose, toxic or infectious neuritis, grippe, syphilis, malaria.	No evident cause (migrainous antecedents frequent). Slow evolution with periods of spontaneous calm growing shorter and shorter.

Lévy is unable to appreciate either the meaning of the term neuralgism, or to see any necessity for its use. He does not favor the oil-injections, believing alcohol to be better.

Treatment. The injection of alcohol for the relief of trigeminal neuralgia is reported in 15 cases by J. A. Bodine and F. C. Keller.[1] From partial to complete relief was afforded in every case, all being very briefly summarized. The method used was that recommended by Lévy and Baudouin, and two cubic centimeters of alcohol more or less diluted comprised the dose. More accurately, the formula was:

Cocain hydrochlorid, 1 grain.
Chloroform, 10 minims.
Alcohol, 3 drachms.
Distilled water, enough to make 4 drachms.

Preliminary anesthesia of the skin at the point of needle entrance with weak cocain solution or plain water, the

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(1) New York Med. Jour., Sept. 26, 1908.

division of the skin by incision with the point of a bistoury, allows easy entrance of the needle point without danger of carrying the infection always resident in the depths of the skin to deeper levels. The details of the method are here omitted, they having been accurately described in the Practical Medical Series, 1907, vol. x., p. 170.

The treatment of neuralgias with alcohol injections forms the title of a paper by W. Alexander,[1] the German literature being fairly well reviewed, but not much space being devoted to French or American writers, with the exception of Lévy and Baudouin, Brissaud and Sicard in France, and Kiliani, Hecht and Patrick in the United States. Alexander concludes that all cases of severity, before operation is recommended, should be treated by alcohol injections; that all mild cases in which hitherto subcutaneous resections have been made, should be at once treated by alcohol injections.

The *surgical treatment* of trigeminal neuralgia is described by J. B. Deaver,[2] and the numerous intracranial causes briefly reviewed, among the latter being fracture of the base of the skull, periostitis, abscess, aneurysm of the internal carotid artery, tuberculosis and syphilitic granulomata, and even tumors of the Gasserian ganglion itself. These cases Deaver believes should be treated surgically, and his method of procedure is set forth.

FACIAL SPASM.

Clinical case. A case of spasm of articulate speech with hemispasm of the face, and bilateral spasm of muscles of the neck and of the shoulder-girdle is reported by L. Rimbaud and J. Anglada[3] in a patient, 31 years of age. The spasms of speech began when the man was 22 or 23 years of age, and in 7 years attained a maximum at which it has remained for 2 years. When asked a question the disturbances begin and attain their maximum in 5 to 10 minutes. The voice is feeble and at times is entirely cut off by an apparent constriction of the glottis. At the same time the muscles of the face and neck contract, then those of the

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(1) Berliner klin. Woch., Nov. 30, 1908.
(2) New York Med. Jour., March 27, 1909.
(3) Nouv. Icon. de la Salpêtrière, March-April, 1909.

shoulder-girdle group. These movements, including the spasms of the occipito-frontalis (wrinkling of the forehead) are shown in Plate X.

Treatment. Facial spasm has been treated by H. T. Patrick[1] by injections of alcohol successfully in three cases. Facial spasm may be distinguished from tic by the following contrasting points:

"1. Tic is much more common than spasm and invariably develops in a nervous or neuropathic individual. Disposition or temperament appears to have nothing to do with spasm.

"2. Facial spasm is a real spasm, pure and simple; an isolated disorder strictly confined to the mechanism of motor innervation of the face. Tic is not a spasm at all but a volitional movement (even when automatic and subconscious) with extensive psychic and sensory associations. It is a motor obsession or rather the motor expression or result of an obsession. Consequently:

"3. Spasm is absolutely devoid of voluntary or involuntary control while tic is always to some extent under the control of the will and always subject to involuntary control by strong emotional or intellectual preoccupation.

"4. To state it another way, spasm may be said to be an anatomical, tic a physiological, disorder. A good picture of facial spasm may be obtained by faradization of the facial nerve. Tic is always the replica of a perfectly natural (though maybe unusual) physiological movement. Looking at the distortion caused by faradization no one would mistake it for voluntary contraction of the facial muscles. On the other hand, having seen only one individual contraction of facial tic, no one could say that the movement was not a perfectly normal and natural one—possibly unusual, possibly overdone, but the legitimate result of a casual cause. Voluntary imitation of facial spasm is practically impossible. The patient himself can always reflect or imitate his tic movements and another person can nearly always do so.

"5. In harmony with the anatomical relations of spasm are the facts that in its incipiency it is confined to *part* of a muscle (generally the orbicularis palpebra-

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(1) Jour. Nerv. and Ment. Dis., Jan., 1909.

rum), that eventually it takes in the entire distribution of the facial nerve, no more and no less, and that however bad it may be it is strictly unilateral. In harmony with the physiological relations of tic are the facts that it never affects part of a facial muscle, because we cannot voluntarily make fascicular contractions, that it rarely includes all the facial muscles in any given moment, that one physiologically associated group of facial muscles may contract one moment, and another group the next, that it is very apt to be bilateral either simultaneously or alternately, and that it is peculiarly liable to be associated with tic of nearby or remote muscle-groups.

"6. The contraction of facial spasm is very like that produced by faradism when the vibrator is not running smoothly. That is, while the general effect is tonic, there is, besides, a flickering or quivering or rapid, slight, irregular twitching such as never occurs in voluntary movement or tic, and cannot be imitated. Each spasm begins with this flickering contraction, generally of part of a muscle and most frequently in the orbicularis palpebrarum. The preliminary quiver may be exceedingly brief or may last several seconds, and even in a fully developed case one often sees these little abortive spasms. Nothing of the kind is seen in tic.

"7. Facial spasm may not look any worse than facial tic, but it is much more uncomfortable than tic, much more of a disability. The subject of tic may talk and sing as he pleases. To be sure his talk will be interspersed with grimaces, but his sentences are not interrupted by them. A patient with spasm may be cut short at any time.

"Injection of the seventh nerve by alcohol for the treatment of facial spasm naturally suggests itself, in view of its successful use in trigeminal neuralgia. The pathology of facial spasm being unknown, no entirely rational treatment is possible. The injection method is empiric, is new and may be found to have disadvantages not now known; but it is exceedingly simple and the results are far better than those of any treatment with which I am acquainted. Of course the great drawback to the treatment is the facial paralysis which it causes, and for this reason the method is scarcely applicable to very mild cases."

The cases reported are three in number, the first case being that of a colored woman, 38 years of age, with a history of spasm extending over 12 or 13 years. Plate XI shows the spasm and cure. The injection was made Sept. 19, 1907, 15 to 20 minims of a 75 per cent. alcoholic solution being used. "On Oct. 2 the patient presented an ordinary peripheral facial paralysis. She had had no signs of spasm except on the third day when she had noticed a very slight twitching about the eye." Even on Oct. 2 the patient could close the left eye. The cervico-facial division, however, seemed to be quite paralyzed.

PERIPHERAL NERVES.

BRACHIAL PLEXUS RADICULITIS.

A case of radicular paralysis of the brachial plexus is reported by Leclerc and Chattot.[1] The patient was a girl, 18 years of age, who had carried a big watering-pot for the three weeks previous to the onset of her trouble, while working for a gardener. The onset was abrupt with severe pain in the right shoulder and neck, followed the next morning on awakening by a paresis of the hand and forearm. Examination showed all the extensors to be involved, and the abductor pollicis was also paretic, while the two adductors and the opponens pollicis were intact. The flexors were very slightly paretic, and so also were the interossei. There was no muscular atrophy anywhere. The muscles of the shoulder and arm, and the supinator longus were intact.

Sensation was profoundly affected. Anesthesia was present in the form of a band, deepening as the end of the upper extremity was reached. It began just below the elbow and extended along the inner border of the forearm, ending in the hand on the hypothenar eminence and the corresponding portion of the palm. The thenar eminence and adjacent palm surface were not affected. On the dorsal side of the hand only the ulnar border was anesthetic. The anesthesia was absolute. Neither touch, the prick of a pin, or hot and cold were felt. Even the muscle-joint

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(1) Lyon Méd., Nov. 29, 1908.

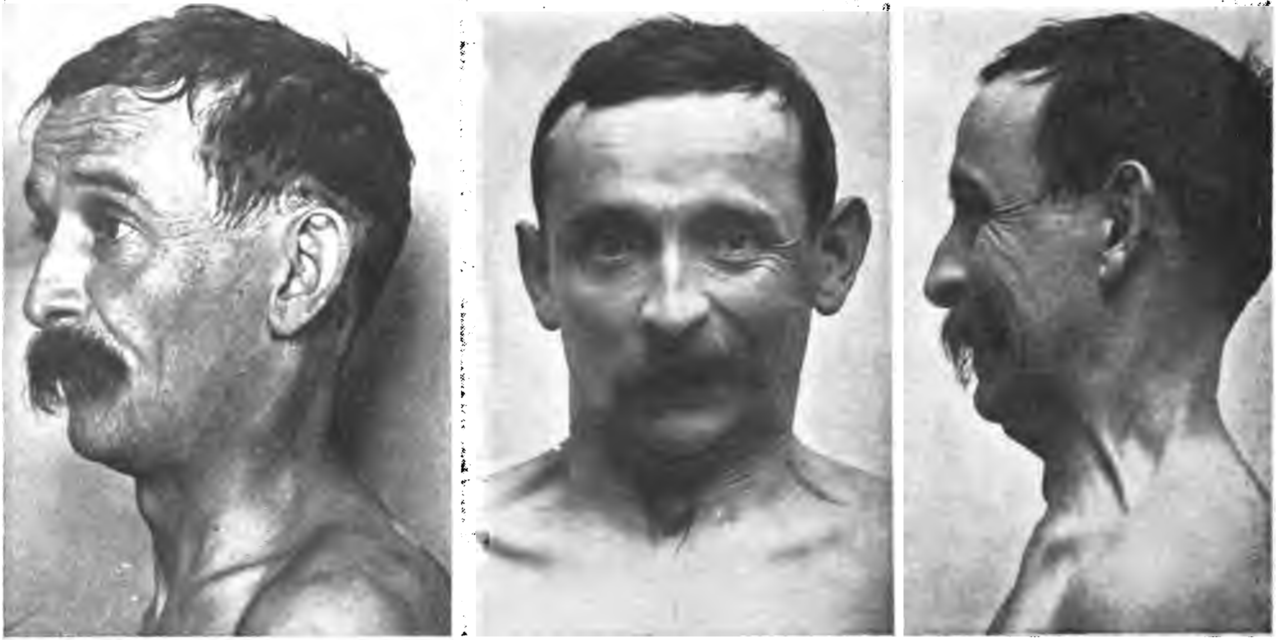
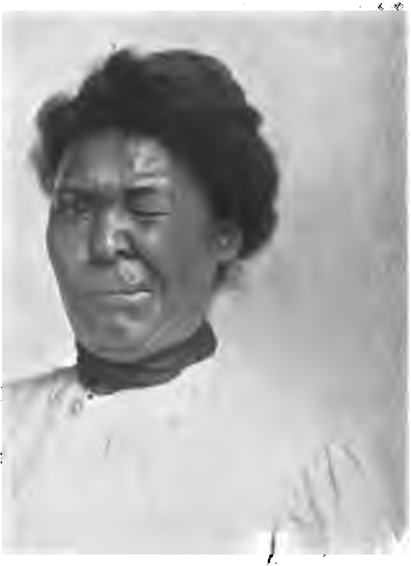
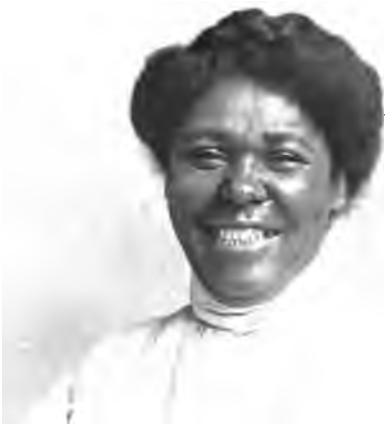


PLATE X.

Facial hemispasm and bilateral spasm of the muscles of the neck and shoulder girdle. Spasm of articulate speech.—Rimbaud and Anglada. (Page 197.)



Typical Facial Spasm.—Patrick. (Page 199.)



Facial Spasm, six and a half months after treatment. Patient smiling to show absence of facial paralysis.

PLATE XI.

sense was abolished, and also the stereognostic sense. There were no reflex disturbances anywhere.

Electrical stimulation of the musculo-spiral gave no response except in the supinator longus, and the long and short radial extensors. The extensor communis digitorum could not be made to contract. The reaction of degeneration was found in the muscles supplied by the posterior interosseous branch of the musculo-spiral nerve. Thus hysteria was eliminated from the diagnosis. Moreover the distribution of the trouble was not wholly attributed to the involvement of any particular branches of the brachial plexus, in view of the fact that both extensors and flexors were involved. The diagnosis of a radicular paralysis seemed the only possible one, traumatic in origin, due to the heavy burden, pulling the arm down from the trunk. Such an etiology has been reported before by Duval and Guillain (1901), also with an abrupt onset. According to the muscles and sensory areas involved in the present case the roots affected were the seventh and eighth cervical, the fifth and sixth being intact (no involvement of the supinator longus or radial extensors). Absence of pupillary disturbances excludes the first dorsal roots.

Leclerc[1] also reports a case of radicular paralysis of the brachial plexus in a case of neurofibromatosis or von Recklinghausen's disease.

MUSCULOSPIRAL PARALYSIS.

Musculospiral (radial) paralysis due to dislocations of the head of the radius forms the title of a thorough paper by DeWitt Stetten.[2] Interest in the subject was aroused in the writer by the case of a printer, 19 years of age, who injured his arm in such a way that he could not extend his wrist or fingers. A diagnosis of fracture of the ulna was made by the physician attending him and the arm was placed in a splint for four weeks. When seen by Stetten three months later, there was wrist-drop, atrophy of the forearm muscles, and a glossy appearance of the skin of the dorsum of the hand. Sensation was lost in the territory of the radial nerve-distribution. Operation was per-

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(1) Lyon Méd., Dec. 20, 1908.
(2) Annals of Surgery, August, 1908.

formed with a view to investigate and repair the nerve-injury if possible, and to increase the flexion of the elbow by either reducing the dislocation or resetting the radial head. The technic of the operation is carefully described. The outcome was very successful.

A study of the literature shows very few similar cases, only 8 being found, and summarized, the author's being the ninth case.

The prognosis of this form of musculospiral paralysis is good; in only two of the cases reviewed was it permanent. Six of the cases were positively cured. Treatment consists primarily in freeing the injured nerve, and suturing the ends, if it is severed. Electricity, massage, active and passive motion are all valuable.

The following conclusions are drawn:

"1. Musculospiral paralysis as a result of dislocation of the head of the radius is a distinct type of nerve injury, and is quite as definite and characteristic as any other form of injury to this nerve. In fact, in every case of anterior dislocation of the head of the radius the two divisions of the musculospiral nerve are in danger and it is a fortunate accident if they escape.

"2. Its actual occurrence is naturally rare, but comparatively, *i. e.,* compared to the frequency of the joint injury itself, it is not infrequent.

"3. It is most likely to occur when the dislocation accompanies ulnar fracture and when the direction of the dislocation is forward and slightly outward.

"4. The nerve is almost invariably injured below its point of division into the posterior interosseous and radial. One or both branches may be caught by the dislocated head.

"5. The symptoms are practically those of the typical musculospiral palsy. They may vary greatly in extent or degree, depending upon whether one or both nerves be injured and how badly. The supinator longus muscle escapes involvement.

"6. The prognosis is good under appropriate treatment, which is practically the same as that for the dislocation: simple reduction, if possible, and generally resection of the head of the radius in old cases, with nerve suture if necessary.

"7. Should the nerve be uninjured in a dislocation of the radial head, its close proximity to the bone should be borne in mind, the dislocation should be reduced, and care should be exercised not to injure the nerve, by pressure, hyperextension or careless operative technic."

PARALYSIS OF THE POSTERIOR THORACIC NERVE.

Paralysis of the serratus magnus muscle is of unusual occurrence, and a case is reported by Reubsaet and Barbier.[1] A coachman fell from his box striking his shoulder, just at the acromioclavicular junction, upon the wheel of the carriage, and for two weeks was laid up with pain in his shoulder and at the base of the neck. He then resumed work. Two months later violent subclavicular pain developed, radiating into the arm, and he was forced to stop work. He could not raise his arm above the horizontal, and when he tried it the shoulder-blade would leave the side of the chest, its lower angle projecting five cm. from the surface of the thorax. Examination showed faradic excitability of the serratus magnus to be almost abolished. Galvanic excitability was diminished, and the reaction sluggish. Moreover the lower part of the trapezius was also diminished in faradic excitability. All of the remaining muscles were normal, except a few fibers in the rhomboidei. Such association has been previously reported. It would indicate that the scapular portion of the trapezius is anatomically and physiologically distinct from the clavicular portion; and that the serratus magnus and thoracic portion of the trapezius form a group of synergic muscles.

SCIATICA.

Two cases of symptomatic sciatica, one due to an osteosarcoma of the sacrum, and the other to tuberculosis, are reported by Lesieur, Froment and Leriche from the services of Lépine and Poncet.[2] In the first case there was a right-sided sciatica without radicular anesthesia, but there was atrophy of the quadriceps and absence of the right knee-jerk. Dorsal decubitus increased the pain.

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(1) Le Progrès Méd., 1909, p. 111.
(2) Lyon Méd., Dec. 20, 1908.

Examination showed a fluctuating mass in the right lumbar region, and rectal examination suggested Pott's disease. The postmortem showed an osteosarcoma involving the last two lumbar vertebrae and the uppermost segment of the sacrum, with a very large secondary abscess. Search of the literature showed about 25 cases of tumor of the sacrum, excluding congenital cases, most of them being osteosarcomata, some being possibly traumatic in origin. In these cases it is not always possible to palpate the tumor by rectum. The pain is very severe, even atrocious, and is aggravated by dorsal decubitus. The backbone is held as rigidly as in Pott's disease. Rarely are there signs of a cauda equina lesion.

The second case with a history of a marked left-sided sciatica four months previously complained of very severe sciatica in the right leg. Very striking was the existence of plantar hypesthesia of the radicular type, immediately suggesting pressure on the cauda from the backbone. Examination of the latter showed a swelling over the sacrum, but without any indication of sacro-coxalgia. Surgical intervention disclosed sacral tuberculosis, and inoculation of a guinea-pig resulted positively. Primary and isolated tuberculosis of the sacrum is very rare; when it occurs it is usually secondary to tuberculosis of the lumbar vertebrae, or to a tubercular sacro-coxalgia. The symptoms usually embrace signs of cauda equina compression as well as sciatica.

Differential Diagnosis. Two points in the differential diagnosis of sciatica are mentioned by S. Erben.[1] (1) Only those cases of suspected sciatica which react with pain on contraction of the abdominal muscles are cases of true sciatica. If upon coughing or sneezing there is no pain in the calf or buttocks, the case is not one of sciatica. Conversely, if pain appears, the case is one of sciatica. Consequently in no case should questioning upon the points of pain in defecation, coughing or sneezing be omitted.

(2) A second point is to be found in the behavior of the lumbar vertebræ in flexion of the spine. These are less movable in the sciatic patient than in the normal individual. Either not all of the spinous processes spring

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(1) Wien. med. Woch., Aug. 28, 1909.

into prominence when the back is bent, or the lumbar vertebrae may not bend at all.

Treatment. The treatment of sciatica by deep perineural infiltrating injections of salt solution is advocated by D. Hecht,[1] based upon his experience with 16 cases. Of the salt solutions any one of three may be endorsed as useful and harmless: (1) Normal salt solution at body temperature injected in the amount of 80 to 120 c. c.; (2) normal salt solution refrigerated to zero centigrade, as suggested by Schlesinger, and injected in the amount of 10 to 20 c. c.; (3) the beta-eucain salt solution at body temperature (Lange), injected in an amount of 60 to 100 cc. Alcohol is dangerous and has caused paralysis with muscular atrophy, so that it is condemned by most writers. Figure 19 illustrates the author's method.

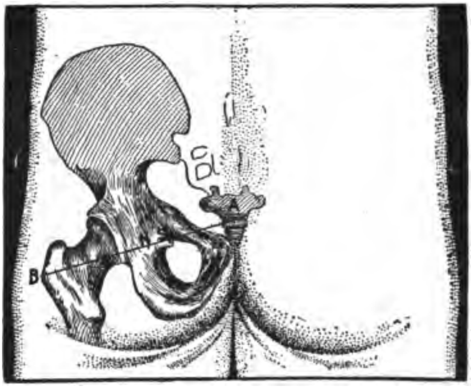

FIGURE 19.

Illustrating landmarks for reaching sciatic nerve at notch. The linear measurement in drawing is necessarily curvo-linear on the body. (a) Sacro-coccygeal joint. (b) Trochanteric border. (s) Spine of ischium. (n) Sciatic nerve a thumb's breadth to outer side of spine. Anatomic accuracy has been somewhat sacrificed to illustrate landmarks. The symphysis and coccyx should not touch when the pelvic bones are tilted at their proper angle.

The treatment of sciatica by continuous extension, long ago advocated in this country, is urged by P. Carnot.[2] Though he has had good results from superficial injections of sterilized air, deep injections of normal salt solution, and stovain, still these remedies are not invariably successful, in which case continuous extension is valuable. An ordinary extension-apparatus is used, a weight of only 5 lbs. being applied. At first extension is continuous, but after a time the weight is removed for longer and longer periods of time during the day, until it is made use of only at night. Sometimes a weight of 5 lbs. may not control the pain, when an increase to 7 lbs. will do so. The mechanism of cure lies wholly in extension of the nerve.

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(1) Jour Am. Med. Assoc., Feb. 6, 1909.
(2) Le Progrès Méd., June 10, 1909.

J. B. James[1] advocates injection of sulphuric ether, combined with either cocain or morphin, 5 m. of the former with 2 or 3 m. of a 1 to 12 solution of either of the latter. The needle used should be 2½ in. long. Unless the patient involuntarily shoots out the leg the nerve has not been touched by the needle. His first case so treated was in 1883.

NEUROFIBROMATOSIS.

Von Recklinghausen's syndrome (neurofibromatosis) associated with tuberculosis is reported by Pic and Gauthier.[2] The authors prefer the term Recklinghausen's syndrome to generalized neurofibromatosis, because the former does not prejudge the nature or extent of the lesions which at best are very variable. The patient, a woman aged 48 years, entered the hospital because of cough and weakness. She gave a history of a cold on the chest from years before, and the usual complaints of a consumptive. She showed all over her body an enormous quantity of cutaneous tumors of various sizes which began to appear when she was 14 or 15 years old. No major or "royal" tumor could be found at first, but in the left tibio-tarsal region and in the region of the heel was a swelling of the consistency of a myxoma which might be so designated. Pigmental patches of a coffee-color were found on the lower abdomen and over the buttocks. Nothing was found on the nerves. The lungs showed signs of tuberculosis.

In this case but two of the three classic symptoms were present: the cutaneous tumors in extraordinary abundance

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(1) British Med. Jour., Oct. 10, 1908.
(2) Lyon Méd., Dec. 27, 1908.

and the pigmental spots. There were no tumors along the nerve-trunks. There were also present marked signs of suprarenal disease: arterial hypotension, asthenia and vasomotor disturbances. The association of von Recklinghausen's disease with the insufficiency of various glands having internal secretions, thyroid, pitiutary, ovarian, and especially suprarenal, has been already noted. Moreover the association of this disease and of tuberculosis has been reported, and the authors suspect that such association is more than a simple coincidence, though they do not affirm it.

Neurofibromatosis with involvement of both peripheral and intracranial nerves is reported without post mortem by W. Healy.[1] The symptoms of intracranial neurofibromatosis are those of tumor of the brain in general plus the findings significant of involvement of special cranial nerves. Two peculiarities are of diagnostic value: (1) There are quite often simultaneous or almost simultaneous signs of nerve involvement on both sides. (2) There are apt to be remissions of months' duration in the symptoms. In Healy's case the second, fifth, sixth, seventh, eighth, ninth and tenth nerves were involved. Deafness was complete in both ears.

MISCELLANEOUS.

TIC AND SPASMODIC TORTICOLLIS.

The relation between tic and spasmodic torticollis is considered by H. T. Patrick,[2] apropos of some remarks on tic and chorea. Personally, Patrick believes spasmodic torticollis to belong with the tics. "If it be not a true tic, certainly the relationship is very close. When Brissaud called it 'mental torticollis' he did a helpful thing in emphasizing its essentially psychic nature. On the other hand it cannot be denied that the disorder in some ways approaches writer's cramp and allied disorders. Still, the points of resemblance with tic are much more striking." The following are mentioned:

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(1) Jour. Am. Med. Assoc., March 20, 1909.
(2) Jour. Am. Med. Assoc., May 1, 1909.

"1. The subject of spasmodic torticollis, as the subject of tic, is always a neuropath. He shows the same sort of abnormal susceptibility, the same psychomotor inability, the same lack of inhibition.

"2. Just as tic ordinarily has a sensory origin, so has spasmodic torticollis. For example, muscular rheumatism, the ordinary 'stiff neck,' is not rarely the origin of the trouble.

"3. The emotional state of the patient has much to do with the neck spasm. It is always worse when the patient is self-conscious or 'upset.' On the other hand intense mental preoccupation may entirely abolish the trouble—for a brief space.

"4. In torticollis there is the same strain in repressing the muscular drawings as in other tics, and the same sense of relief in 'letting it go.' A patient riding in a street car will repress the movements until he can stand it no longer. Then he will hide behind his newspaper and have a little motor indulgence—which gives him a measure of sensory satisfaction or relief.

"5. The muscular contractions of spasmodic torticollis are not true spasms; they are volitional. And they always are more or less under voluntary control.

"6. The patient with spasmodic torticollis invariably has some trick of restraint: what Brissaud has called a restraining gesture. It is one of the most striking things connected with this peculiar neurosis. A man whose neck muscles are twisting his head around with the greatest violence will gently place a finger against his chin or lay his palm over the occiput, when the muscles at once relax and the head assumes its normal position. Oftentimes if the head can merely touch the back of a chair or the wall the trouble is suspended."

RAYNAUD'S DISEASE.

B. Sachs[1] believes that Raynaud's disease, erythromelalgia, acrocyanosis chronica and the conditions allied to them, far from being trophoneuroses, are to be looked upon as pure angioneuroses, very occasionally due to some associated central nervous affection, most usually due to some

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(1) Amer. Jour. Med. Sciences, October, 1908.

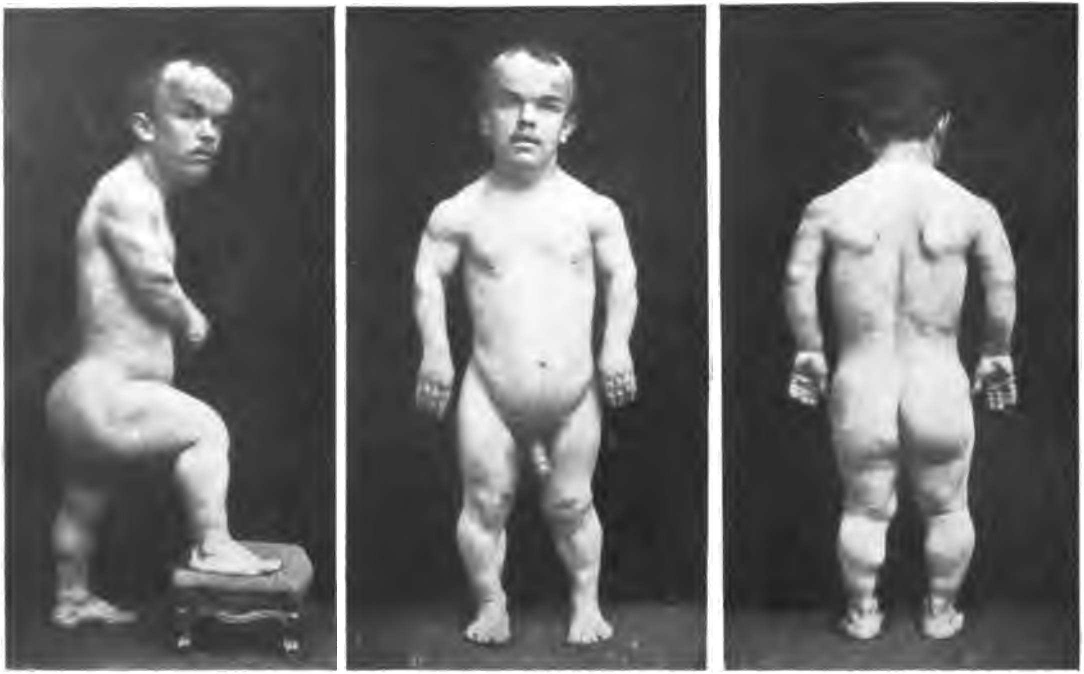

PLATE XII.

Achondroplasia.—E Levi. (Page 210.)

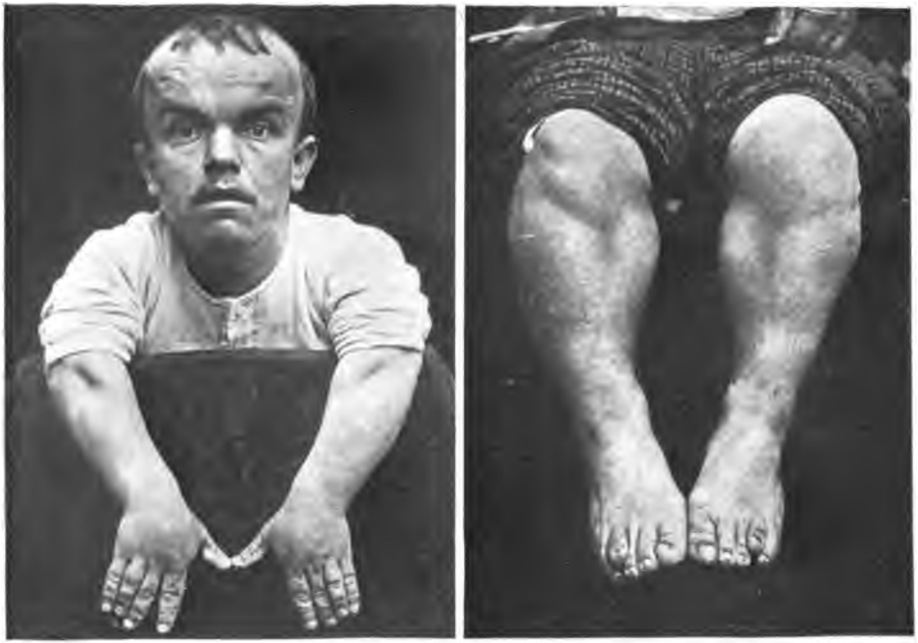

PLATE XIII.

Achondroplasia.—E Levi. (Page 210.)

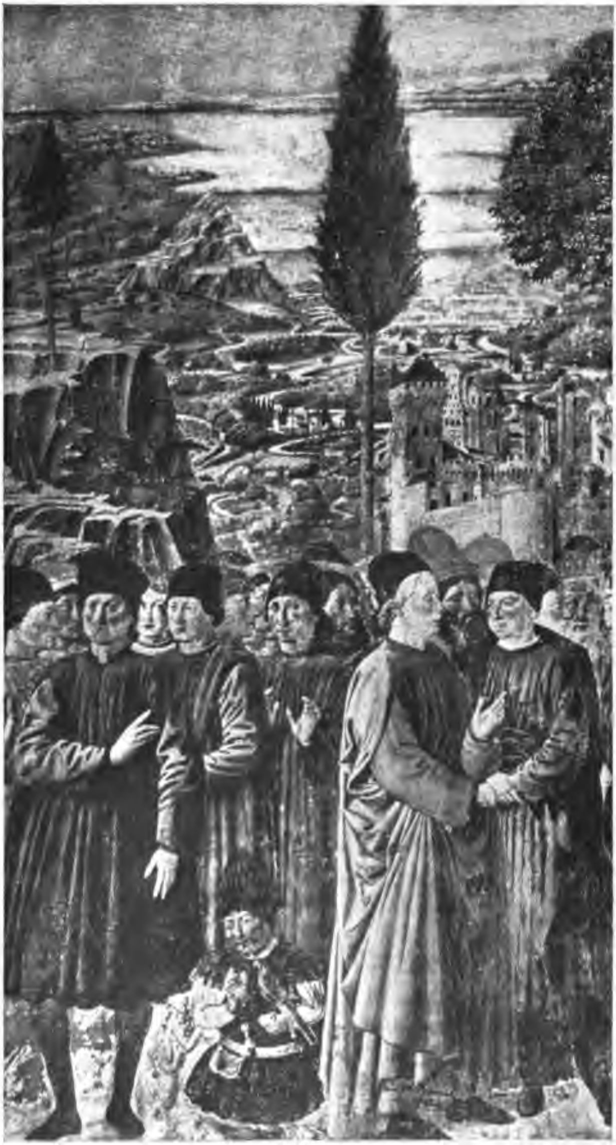

PLATE XIV.

An achondroplasic dwarf.
Fragment of a fresco of Benozzo Gozzoli, Campo Santo of Piso.—
E. Levi. (Page 210.)

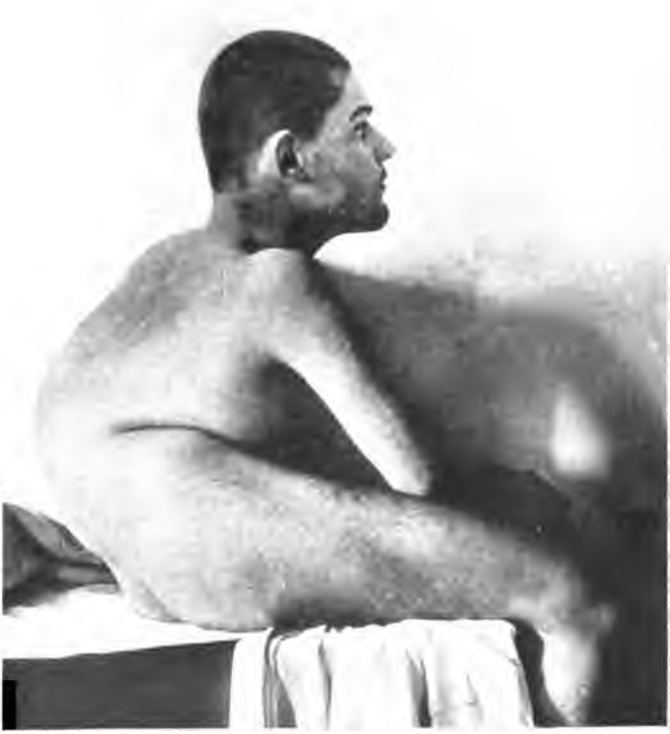

PLATE XV.

Primary myopathy.—Vires and Anglada. (Page 212.)

independent form of vascular disease. Experience has taught him, for example, that very few cases of erythromelalgia are associated with spinal symptoms. So far as the author's hospital cases are concerned "it is probable that malnutrition, premature arteriosclerosis, and the peculiar occupations (tailoring, sewing machine work—all of them involving a retardation of the venous circulation) are responsible for the frequent occurrence of these special disorders." Pathologic studies demonstrate that in all of these allied disorders there is a striking interference with the peripheral blood-supply, which is much the same anatomically, whether the cases be designated clinically as erythromelalgia, Raynaud's disease, or acrocyanosis.

In summarizing his clinical experiences, many of them substantiated by pathologic studies by Buerger of some portions of diseased amputated extremities, Sachs makes the following points:

"1. Erythromelalgia and Raynaud's disease are often associated with one another; a sharp line of demarcation between the two cannot always be maintained; and the types blend into one another so readily that the exact labelling of a case is often a matter of great difficulty.

"2. The clinical symptoms at one part of the disease correspond closely to the erythromelalgic type, and at a later stage to the Raynaud type.

"3. Erythromelalgia is relatively frequently associated with gangrene, generally dry gangrene.

"4. There are cases which, in their incipiency, present typical symptoms of Raynaud's disease, and as they progress might very well be designated cases of spontaneous gangrene due to obliterating endarteritis (or angiothrombosis—Buerger)."

The case-histories of six patients are appended in illustration. Special attention is directed to the association of intermittent claudication with symptoms of the Raynaud type, and a case is reported in which a distinct intermittent claudication is associated with undoubted evidence of disturbances in the peripheral circulation of both the hands and feet, in every way resembling the symptoms of Raynaud's disease.

A case of Raynaud's disease simulating Morvan's disease is reported by Mouisset and Chalier.[1] The patient, a man 39 years of age, entered the hospital on account of suppurations involving the ends of several of his fingers. While in the military service in Indo-China he had malaria, and it persisted seven or eight years after his return to France. For five years he has been free from it. His present illness began five years ago with a painful whitlow on the ring-finger of the left hand. Subsequently similar suppurations appeared on all the other fingers except the little fingers, and also upon the left thumb. They were very sluggish, lasting from a few weeks to five or six months, and the pain often extended up the forearm and arm, even to the shoulder. Frequently there appeared, especially in winter, states of syncope and asphyxia in the fingers, as in all well-defined cases of Raynaud's disease. The toes, nose, and ears were never affected. The dissociation symptom of syringomyelia was negative. In Morvan's disease, too, the whitlows develop without pain.

ACHONDROPLASIA.

A case of achondroplasia in an adult is described by E. Levi, and is illustrated by plates XII, XIII and XIV, the latter being from a fresco in Italy.[2] Thus far about 50 cases have been reported in the literature. Achondroplasia has been regarded by some as the opposite to giantism, and has therefore been ascribed to certain glands with internal secretions. The patient here reported is a man 25 years of age. He was born at term, but from his birth his mother noticed that he had a very large head and very short arms and legs. He developed normally, began to walk early, and at two years of age spoke correctly. His dentition was also normal. He was brought up as a public charge and learned to read and write very early. During boyhood the disproportion between trunk and limbs became accentuated. Sexually he developed normally, and even masturbated as a boy. The plates illustrate better than words his general appearance, his height being 3 feet 9 inches, and his weight being 78.5 lbs. The di-

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(1) Lyon Méd., Jan. 31, 1909.
(2) Nouv. Icon. de la Salpêtrière, March-April, 1909.

mensions of the trunk were approximately normal, the distance from his epigastric angle to the pubis being 18 inches, while the distance from the pubis to the floor when he stands, is only 16.8 inches.

Study as to possible etiology showed nothing. The blood was normal, the urine showed as its only abnormality an increase in indican; and the sero-reaction of Wassermann (deviation of the complement) was completely negative. Histologically these cases show a sclerosis of the epiphyseal cartilages, of a type often seen in infections, especially bacterial, alcoholic, etc. The authors believe in the infectious hypothesis as contrasted with the autointoxication theory for the cartilage sclerosis enunciated by P. Marie in the report of his case in 1900. As to the question of the influence of the ductless glands upon the production of this condition, the authors are unable to see how the pituitary gland can be at fault in these cases.

GENERALIZED MYOPATHY.

Generalized myopathy with pseudohypertrophy and atrophy in an individual observed over a period of ten years is reported by Vires and Anglada.[1] On neither the paternal nor the maternal side was there bad heredity. Up to 7½ years of age the patient was perfectly healthy, but at this time he fell into a ditch full of water, and remained wet through for hours. Shortly after it was observed that he was becoming clumsy, that he ran with difficulty and frequently fell down. It seemed to be hard for him to climb stairs, and it was noted that he clung to the balustrade. Weakness became generalized very rapidly, but the calves remained as large as ever. Examined early in 1902, he showed a slight skoliosis, with the convexity to the right, the shoulderblades were prominent, the lumbar muscles were atrophic, but the buttocks appeared of normal size. The arms were slender, the forearms atrophic, the thenar and hypothenar eminences flattened. The interosseous spaces were normal. The lower extremities showed a remarkable contrast between the emaciated thighs and the hypertrophied calves. There was a bilateral

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(1) Nouv. Icon. de la Salpêtrière, May-June, 1909.

toe-drop. Walking was difficult. The electric examination gave a slightly-delayed reaction in the calves. The skin nowhere showed sensory or trophic disturbances. Walking became worse, and since 1905 has been impossible.

At present, 9½ years after the onset of his illness, the patient, now 16½ years of age (see Plate XV) shows a flattening of the head from front to back, and a widening transversely. The head has a tendency to fall backward, the neck is down between the shoulders, which are thin and prominent. The shoulder-girdle and arm muscles are wasted, the head of the humerus appearing beneath the skin. The trunk is as it were telescoped upon itself, there being an extreme dorso-lumbar skoliosis. The thighs are hypertrophied, and are kept semiflexed upon the pelvis, as the patient lies in bed. The feet show the equine type. Mobility is greatly reduced, walking or even standing being impossible. The patellar reflexes and the Achilles-jerks are abolished; the cremasteric reflex remains.

Very interesting is the cardiac hypertrophy, not found in 1902. The apex is in the seventh interspace beyond the left nipple-line; the apex-beat is energetic, but there are no abnormal sounds or murmurs over the cardiac valves. A slight systolic murmur was noted over the valve of the pulmonary artery, however.

A very unusual association of findings is reported in a case shown by Ziehen[1] at the Charity Hospital in Berlin. The patient, a lad of 18 years of age, began to walk when he was 5 years old, and to talk when he was 2½ years of age. Examination disclosed a complete ophthalmoplegia externa, a congenital diplegia facialis, atrophy of the right half of the tongue, and complete absence of the left pectoralis major. The latter is quite unusual, about 100 cases only being mentioned in medical literature. The interest in this case lies in the fact, not of the defect of the pectoralis major, but in the association of that defect with ophthalmoplegia externa, facial diplegia, and atrophy of one-half of the tongue.

After reporting the case carefully Ziehen reviews these cases in general, and then offers the following table for their classification:

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(1) Berliner klin. Woch., Aug. 24, 1908.

(A) Congenital atrophic paralyses or defects:
  (a) Nuclear;
  (b) Primary muscular defects.
(B) Acquired atrophic paralyses based upon a congenital predisposition:
  (a) Nuclear; infantile progressive bulbar paralysis;
  (b) Primary myopathia; dystrophia musculorum progressiva.

Group (A) may be separated from group (B) by the fact that the symptoms in the former are never progressive, whereas in the latter they are. The former cases are all due to aplasias or hypoplasias, whereas the cases in group (B) are due to the gradual repression of nuclei or muscles, and are hence progressive.

Ziehen also believes that the sub-groups (a) and (b), nuclear and muscular respectively, may be differentiated by electrical excitability. If there is a primary muscle-defect, the electric excitability is completely lost, or quantitatively much reduced. If there is a nuclear defect, not only in the electric excitability reduced quantitatively, but in at least some bundles, a slight or pronounced degenerative reaction is found present. Only in those cases where the muscle defect is total is there inability to distinguish by this means a complete muscular aplasia from a nuclear aplasia. But in hypoplasia, the nuclear form may be distinguished from the muscular by the presence of fibrillary tremor in the former and its absence in the latter.

In the case reported Ziehen shows a lack of progression, which fact places it in group A, consisting of the congenital atrophic paralyses. The patient showed fibrillary tremor of the tongue, and partial electrical reactions of degeneration in the face, thus placing him in subgroup (a), those of nuclear origin. Hence in this case we must suppose congenital nuclear defects in the nuclei of the motor oculi, pathetic, and abducens, the facial, and the centers for the pectoralis major.

MYATONIA CONGENITA.

Congenital muscular atony form the title head of an extensive review by Lévi-Sirugue.[1] The first notice of this malady came from Oppenheim in 1900, who gave as its characteristics (1) its appearance at the beginning of life, (2) the absence of any hereditary or familial character, (3) a regressive evolution, and (4) loss of tonicity in the muscles which are not paralyzed. He proposed the term *myatonia congenita,* which has been generally adopted. A bibliography of the known cases is given, including that of Carey Coombs, reported in the Practical Medicine Series, 1907, Volume X, p. 203, and Leclerc's case, reported in the Practical Medicine Series, 1908, Vol. X, p. 215.

Lévi-Sirugue thoroughly discusses the symptomatology. All the limbs are limp and motionless, due to a remarkable hypotonia or atonia. By reason of the atonia the limbs can be moved to an excessive degree. In the majority of cases such atonia has been noted by the parents from the birth of the child. The arms are ordinarily the only members used, the legs being inert, though they will move when pinched. The muscles as a rule not involved are the eye-muscles, those of sucking and swallowing, the sphincters, and the muscles of respiration, but even some of these have been reported as having been affected. Though the muscles are atonic, there is no atrophy. The knee-jerks are usually absent. Electrical and mechanical irritability of the muscles is intact. No disturbances of sensation exist.

As a rule the cases gradually improve, especially in the arms. Some cases result fatally by reason of pulmonary complications in breathing, or from broncho-pneumonia. The *diagnosis* is easy, but care must be taken not to confound it with acute anterior poliomyelitis, many symptoms of which are found in myatonia congenita. It should also be differentiated from *dystrophia musculorum progressiva,* the paralyses associated with hematomyelia, myelitis, gliosis, or polyneuritis, and *myasthenia gravis.*

Its *pathogenesis* is unknown, one theory being that the muscles fail to acquire their natural tone on account of the arrest of development of the medullo-cerebellar pathways; the other being that the thymus is diseased. Spiller's case showed changes in the thymus. The *treatment* is wholly symptomatic, deep faradisation being advised for long periods of time. Baths and massage complete the list of therapeutic agents of any value in its control. Many cases improve spontaneously. Among drugs arsenic and strychnin are recommended.

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(1) Gaz. des Hôp., Feb. 6, 1909.

MYASTHENIA GRAVIS.

A very thorough and able review of the literature of myasthenia gravis, together with the report of two cases, including the post mortem findings of one, comes from the pen of G. Marinesco.[1] In his review of the literature it is gratifying to Americans to note how much attention is paid to their contributions. The historical review is very complete, the case of Sir Samuel Wilks in 1877 being the first one noted, and 3 others by Erb in 1879. But little attention to these early cases was paid until in 1887 Oppenheim again called attention to the disease, giving it the name of myasthenia gravis. In 1892 Hoppe, a pupil of Oppenheim's, gave as the essential symptoms of the malady: (1) Absence of atrophy and of disturbance of electrical irritability; (2) participation on the part of the upper part of the facial and of the motor oculi nerves; (3) conservation of the muscles innervated by the hypoglossal nerves; (4) marked remissions and rapid variations in the intensity of the symptoms; (5) negative post mortem findings. To these symptoms but two noteworthy additions have ever been made, one in 1893 by Goldflam, who observed that the essential characteristic of the disease is not a paralysis but a rapidly appearing fatigue on the part of the muscles when used; the second by Jolly, in 1894, who discovered the so-called myasthenic muscular reaction which he added to the symptomatology, and which led him to propose the name *myasthenia gravis pseudoparalytica.* The fundamental point in the disease is the fatigue. Rest more or less prolonged restores to a muscle the ability to contract once more for a few times. The muscles earliest affected are those used the most, as the eye-muscles. The tendon-reflexes may show fatigue phenomena. Even sensory centers seem to show the fatigue.

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(1) La Semaine Méd., Sept. 2, 1908.

Most interesting is the pathology. Erb in 1879 concluded that the disease was really a chronic superior and inferior polioencephalitis, and this idea has been supported by many. By Oppenheim, Strümpell, Jolly and others this idea was combatted. The first essential contribution was made in 1901 by Weigert (see Practical Medicine Series, 1902, V. X, p. 203), who found a thyroid containing anatomic elements like those of the thymus, and in the perimysium and between the muscle-fibers masses of cells identical with those in the thyroid tumor. The muscle cell-masses were accordingly supposed to be metastatic manifestations. Since then many other observations have been made in which the muscles have been found at fault. For example, Hun's case in the United States is cited, and many others in the literature. There are in fact three theories as to pathogenesis at the present time: (1) The cortical theory, ascribing the disease to the cerebral cortex, though as Oppenheim has pointed out, mental symptoms are never present; (2) the bulbar theory, ascribing the disease, as did Erb in 1879, to the motor bulbar nuclei; (3) and the muscular theory, ascribing the symptoms to changes in the muscles themselves. This last theory has several subdivisions, the cleverest explanation of the pathogenesis being that of Chvostek, who refers the disease to certain glands with internal secretions which act secondarily upon the muscles in an adverse manner.

Marinesco relates two cases occurring in sisters, the first cases of the sort recorded. Both began with a ptosis coming on at first only when the eyes were fatigued, then disappearing four or five days together, only to reappear, and each time a little worse than the time before. Subsequently in each the face began to be involved, and gradually the disease more and more developed. The older of the two sisters died of the disease, and a post mortem examination was made. Aside from tuberculosis of the right lung there was found the persistence of the thymus and hypertrophy of the hypophysis, besides the changes in the muscles. The latter showed fatty infiltration, multiplication of the nuclei of the sarcolemma, and unaccustomed pallor of a certain number of muscle-fibers. The infiltration of fat did not seem to disorganize the structure of the muscle-fiber. This infiltration Marinesco is not inclined to attribute to a chemical alteration of the protoplasm, but rather to an incomplete combustion on account of insufficiency of oxygen during their functioning. The hypophysis showed pale regions alternating with regions staining well, and as a whole was enlarged. The cells themselves were modified, appearing spongy, and looking like eosinophiles containing colloid matter. The thymus did not present any signs of involution or degeneration, but neither was it normal in structure. The thyroid and the suprarenal bodies also showed changes.

Believing from these findings that myasthenia gravis depends upon a disturbance of oxidation, Marinesco produced by an Esmarck's bandage ischaemia in a healthy patient's arm for half an hour, and then applied an electric current. The myasthenic reaction of Jolly was obtained. Researches made further showed that resistance to fatigue on the part of the muscles is greatly diminished in myasthenia. Possibly something circulates in the blood which causes quick fatigue in the muscles, akin to Weichardt's "fatigue toxin," which he found in the blood. Kauffmann's researches apparently prove that during muscular activity there is in myasthenia a disturbance of oxidation leading to the formation of intermediate acids.

Marinesco concludes thus: "Myasthenia is then a syndrome occurring in an affection of the vascular system of glands which are all so related to one another that an alteration in one brings about an alteration in the remainder. Myasthenia may be considered as the opposite of tetany; and just as tetany can be produced experimentally by the removal of the parathyroid glands, so myasthenia gravis can be produced experimentally by the removal of the suprarenal bodies. While suprarenal extracts are vasoconstrictors and excite a condition of hypoglobulism, so those of the parathyroid are vasodilators and excite a condition of hyperglobulism.

Myasthenia is characterized clinically by fatigue and exhaustion in voluntary movements, with predominance in the muscles acting continually; it is characterized chemically by insufficiency of oxidation; pathologically, by alteration in the striated muscles, and in several glar with internal secretions, such as the parathyroid glands, the liver, suprarenal capsules, and hypophysis. The latter microscopically look like glands in hyperfunction. The prime mover in the disease appears to be a disturbance in the formation of fatigue antibodies or of oxydases, special ferments promoting oxidation.

ADIPOSIS DOLOROSA.

Adiposis dolorosa in a woman, 49 years of age, is reported by T. Klingmann.[1] This disease is closely allied to acromegaly, myxedema, and exophthalmic goitre. The chief complaint in the present case was pain in the left axilla and the posterior part of the left thigh, where there were distinct swellings. Her arms and legs felt heavy and occasionally numb, and she had a feeling of general exhaustion. She weighed 280 lbs., and very painful fatty tumors existed in various places. In conclusion the author states that "adiposis dolorosa is a neuropathic condition of unknown cause occurring in women with neuropathic heredity, and is characterized by painful, fatty swellings occurring in various parts of the body, which condition is preceded or followed by asthenia. Many other symptoms varying from a slight functional disarrangement to organic degeneration are incidental expressions of an inherited unstable nervous system occurring in many other nervous disorders."

A case of adiposis dolorosa with involvement of the large nerve-trunks is reported by P. N. Bergeron[2] in a woman 45 years of age, weighing 175 lbs. She showed circumscribed accumulations of adipose tissue in the bursae about the elbows, and at two of the metacarpophalangeal joints of the right hand. The skin was dry, the subcutaneous fat very sensitive, the reflexes were all diminished, there was marked asthenia without atrophy. Noteworthy points were marked pain over the nerve-trunks on pressure, the sensitiveness of the subcutaneous fat, and the absence of lipomata.

Two cases of adiposis dolorosa with post mortem findings are reported by Geo. E. Price,[3] and are made the basis

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(1) N. Y. Med. Jour., Nov. 14, 1908.
(2) New York Med. Jour., Nov. 28, 1908.
(3) Amer. Jour. Med. Sciences, May, 1909.

of a clinical and pathological study of the disease. Nothing is specially unusual in the symptoms of the cases reported, but inasmuch as but six cases have thus far been examined post mortem, the findings in these two cases are here presented. In the first case, a woman 57 years old, there was found a marked increase of connective tissue in the right lobe of the thyroid gland, without papillomatous ingrowths or plications as described in Dercum's case. In both lobes were many areas of round-celled infiltration, not infrequently filling several adjacent acini. A few hemorrhagic areas were also observed. The hypophysis showed microscopically areas of round-cell infiltration in the glandular portion, and many cells were degenerated. The adrenals were normal.

The second case was a female, aged 48 years, who had suffered with pains in her legs for ten year previous to her death, and who had always been very large. The autopsy revealed, among other features, an abnormally large thyroid gland, with pathologic changes. The pituitary body showed changes similar to those found in the first case. The fatty tissue revealed only here and there some increase in the connective tissue trabeculæ. Separate lipomas were not found. Sections of the radial nerve showed no distinct changes. However, here and there some thickening of the endoneurium existed. The perineurium appeared to be normal. Distinct inflammatory changes were not present.

Summaries of the other six cases in which post mortem findings have been reported are then briefly given.

"The thyroid, affected in seven of the eight cases, at once commands our attention. Considered from the standpoint of the symptoms, however, there is a peculiar contradiction. The obesity, anidrosis, and improvement following the administration of thyroid extract suggest hypothyroidation, while the flushing, tachycardia, tremor, and often the anxious mental state suggest hyperthyroidation. The term dysthyroidismus would seem, therefore, a better one than hypothyroidismus as used by Pineles.

"We should not, however, in considering the pathogenesis of adiposis dolorosa, neglect the hypophysis. It was found distinctly affected in five of the eight cases, and had been unexamined microscopically in at least two of the remaining three cases. It seems, therefore, that sufficient attention has not been given the hypophysis, and I would suggest that etiologically it is of almost as much importance as the thyroid gland. May not the symptom group of adiposis dolorosa result from primary disease of either of these structures, the other being involved secondarily through their close interrelation?"

Treatment. The present status of the treatment of adiposis is uncertain and unsatisfactory. Aspirin, combined with bromid salt will relieve pain. Thyroid gland extract, while not a specific, is the most valuable remedy at hand.

ACROMEGALY.

A post-mortem examination of a case of acromegaly is reported by A. Campbell Geddes,[1] the case during life having been reported by G. A. Gibson.[2] The post mortem findings are detailed at great length, the accompanying Plate XVI showing the enlarged pituitary body, and the skull after removal of the soft tissues of the face.

Macroscopically the following changes were observed: The pituitary body was thirty times its normal size. The thyroid body was ten times its normal size. The parathyroids were largely increased in size; a conservative estimate places the amount of parathyroid tissue at ten times the normal. The thymus was represented by numerous small aggregations of thymic cells. These areas were very vascular. The amount of suprarenal tissue showed a 50% increase. The ovaries were wholly functionless. [Age of patient at death, 50 years.] The spleen was slightly increased. The alimentary canal, respiratory, circulatory, genito-urinary and muscular systems were practically normal. The brain was increased in mass, and the lower part of the medulla and upper part of the cervical cord were compressed antero-posteriorly by bony changes due to acromegaly in the upper cervical vertebræ. The spinal nerves were greatly increased in diameter and length. The sympathetic system was markedly enlarged. The pituitary body was the seat of a simple hyperplasia. The

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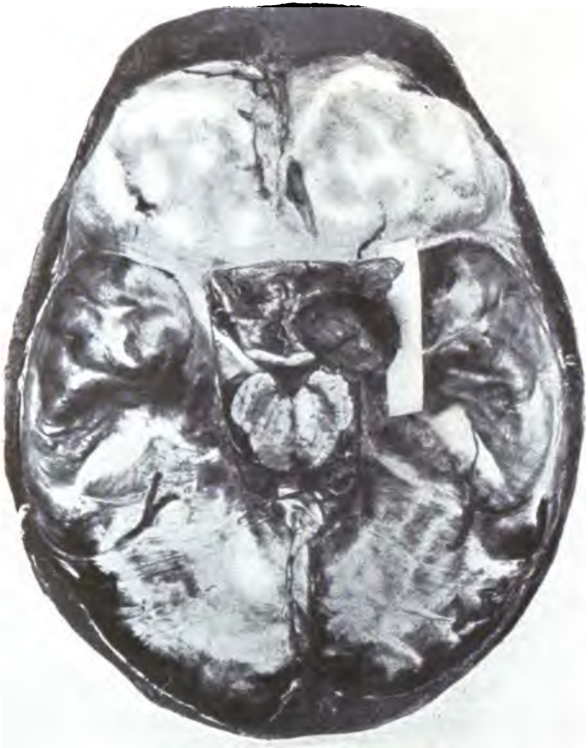
(1) Edinburgh Med. Jour., March, 1909.
(2) Edinburgh Med. Jour., December, 1899.



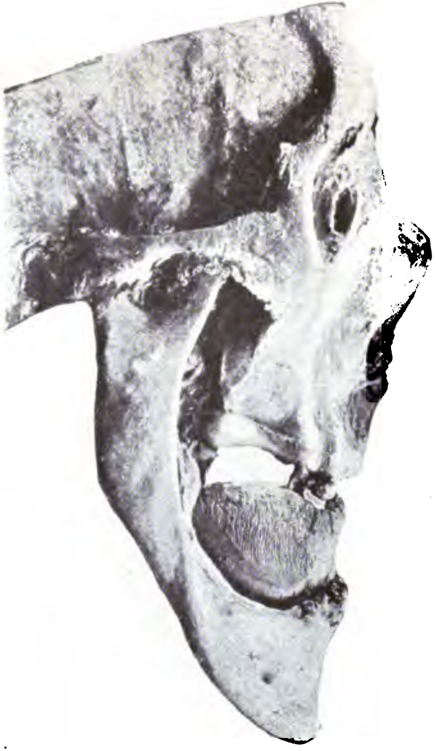


PLATE XVI.

Fig. a.—The interior of the skull after removal of the cerebral hemispheres. A piece of paper has been placed under the edge of the pituitary tumor.—A. Campbell Geddes. (Page 220.)

Fig. b.—The skull after removal of the soft tissues of the face. The plate shows the form of the lower jaw and the mass of the tongue.

thyroid and parathyroids were apparently hyperfunctionating.

Giuseppe Franchini[1] reports an elaborate series of observations on the chemistry and histology of the blood in acromegaly. He inclines to the theory that acromegaly depends upon a perverted metabolism with consequent intoxication. Hence his study of the blood, from which he draws the following conclusions:

"(1) In acromegaly there are frequently found, if not in all cases at least in numerous ones, changes in the blood, especially involving the leucocytes (eosinophilia and changes in the polynuclear leucocytes).

"(2) Chemical changes also occur in the composition of the blood, especially a certain degree of lipemia, and in general, an increase in the mineral constituents.

"(3) That these changes are constant I am not inclined to maintain, because I am convinced that in metabolism as well as in the blood, the changes found cannot be attributed to one type or form."

John Phillips[2] reports a case of acromegaly with thrombophlebitis of the superficial veins, and reviews the literature concerning the malady.

OSTEITIS DEFORMANS OR PAGET'S DISEASE.

A case of osteitis deformans or Paget's disease (see Practical Medicine Series, 1905, Vol. X, p. 216) is reported by B. Pescarolo and M. Bertolotti.[3] The patient, now 53 years of age, had a healthy past except for a few attacks of gout, until he was 38 years old, when he experienced a painful sensation through all of his bones, especially those of the legs. At the same time the small joints of the hands and feet began to be sore, and the dorso-lumbar region of the spine troubled him. These symptoms were at the time ascribed to gout, but treatment was ineffectual. Later other and more alarming symptoms followed; walking became painful, and his legs began to be deformed. Some time later he noticed that his back was getting arched and that he was shorter than he used to be. Then he observed that his hats were becoming too tight, so that each time he bought a new one he had to get one of a larger size. This deformity of the skull came on rapidly and was accompanied by a rapid falling out of the hair and marked tortuosity of the arteries of the temporal and parietal region. (See Plate XVII.) His nose began to turn a little to the left, his chin became prominent and angular, and he found it difficult to chew because his lower jaw seemed to be displaced forward. Yet his general condition as to health remained good. Later gastrointestinal troubles appeared, with attacks of diarrhea. His face assumed the appearance of suffering, his skin became pale and sallow, his head became very broad, and seemed to have taken on an excessive development at the expense of his face. Plate XVIII shows the outline of the trunk and limbs better than any words can depict them. The thorax was deformed, flattened along the sides, the clavicles, scapulæ, and sternum being hypertrophied, but showing no hyperostoses. The bones of the pelvic girdle were also changed, and the anteroposterior diameter was shortened. The legs showed marked curvatures, especially the tibiæ, the height being reduced.

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(1) Berliner klin. Woch., Sept. 7, 1908.
(2) Med. Rec., Feb. 20, 1909.
(3) Nouv. Icon. de la Salpêtrière, May-June, 1909.

These changes in the osseous system were accompanied (1) by cardiac disturbances and dilatation of the heart; (2) by circulatory disturbances characterized by pronounced arterial hypotension and by the appearance of arterial anastomoses very considerably developed in both temporal regions; (3) by disturbances of metabolism revealed by the analysis of the urine and showing a considerable diminution in the elimination of the earthy phosphates.

Paget's Disease in a woman is reported by Klippel and Pierre-Weil, together with a discussion as to its pathogenesis.[1] The patient was a woman 56 years of age, the onset having been at the age of 45 years. The evolution was progressive, painless, but accompanied by some very vague thoracic discomfort. The remarkable point about the case shown excellently in Plate XIX is the unilateral character of the malady. The hand on the affected side shows Heberden's nodes, especially on the index finger, and the phalanges are notably hypertrophied. Yet there is no articular disturbance in the hand or phalangeal joints. On the affected side there is a local hyperthermia, very marked, the thermometer showing as much as 5° C. (9° F.).

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(1) Nouv. Icon. de la Salpêtrière, January-February, 1909.

The authors then discuss the pathogenesis of the disease, but reject for sufficient reasons, one after the other, the theory of syphilis, the trophoneurotic theory, and the vascular theory. The real pathogenesis is as yet not discovered.

TROPHEDEMA.

Chronic trophedema still continues to hold its interest among French physicians. L. Nové-Josserand and C. Laurent[1] have reviewed the subject fairly carefully, without adding much that is new. According to these observers cases can be divided into congenital and acquired, and each of these may be subdivided into familial and individual. The etiology is unknown, the non-familial cases of congenital chronic trophedema being too numerous to permit of any theory of heredity for all cases. As to sex, more females are affected, and the onset of most cases, not traumatic, is between 13 and 18 years in the hereditary cases. In the acquired cases, an antecedent infection has been often noted, as well as trauma. Examination microscopically shows an increase in the connective tissue beneath the skin. It has been thought to be due to some thyroid disturbance, and also to some spinal cord involvement. The traumatic origin is certain in some cases, and it has been thought that the trauma creates a neuritis of the ascending sensory branches, which in turn leads to the trophic disturbance of the subcutaneous connective tissue.

[In this connection may be mentioned the personal case of one of the Editors. His right leg was crushed across the thigh, being caught between the frame of an automobile and a hard object. A large hematoma at once formed, and the thigh became anesthetic in the whole territory of the middle cutaneous branches of the anterior crural nerve. Part of the territory of supply of the external cutaneous nerve of the lumbar plexus, and of the crural branch of the genito-crural nerve, and of the internal cutaneous branch of the anterior crural was also at

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(1) Gaz. des Hôp., April 10, 1909.

once rendered anesthetic to all forms of sensation. A collection of serum formed within ten days' time and ʒxvi was withdrawn on one occasion and ʒxviiss on another, tapping through the anesthetic skin being attended with no pain whatever. There has since developed a trophedema, the injured leg being much larger than the sound one, and showing a little below the groin an especially large roll of hypertrophied connective tissue. Bearing upon the nerve-origin of trophedema in trauma is the fact that sensation a year after the accident has not returned to the thigh. Protopathic sensation has fully returned, but epicritic sensation has not, it being impossible to localize spots when touched anywhere on the whole anterior aspect of the thigh. Though so much damage was done to the cutaneous sensory nerves, not a motor branch or branchlet of the anterior crural (or femoral) nerve was injured, there being not a trace of paralysis.—Ed.]

The cases of trophedema show the edema to be firm, not pitting, and not exuding when scarified. The skin preserves its usual color, the local temperature is not modified, and sensibility is normal. The reflexes are unchanged or diminished. The volume of the edema is occasionally considerable. No symptoms other than the local ones are found. The cases may hardly be said to have an evolution; the edemas appearing quickly, attaining their maximum and there remaining indefinitely. The *treatment* is practically totally inefficient. Massage, electricity, elastic compression, local blood-letting have all been tried. Thyroid extract has proved to be useless.

A case of acquired trophedema in a woman whose ovaries had been removed, who was afflicted with a goitre, and whose mind was lost, is reported by J. Ramadier and L. Marchand.[1] The case may be thus summarized: In a patient whose ovaries had been removed there appeared simultaneously mental disturbances, and a goitre with trophic alteration of the skin, hair, and teeth, and edema of the lower extremities. A few months later the goitre disappeared, but the mental trouble persisted, while the edema of the lower extremities assumed monstrous proportions. (See Plate XX.) The mental disturbances con-

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(1) Nouv. Icon. de la Salpêtrière, May-June, 1909.

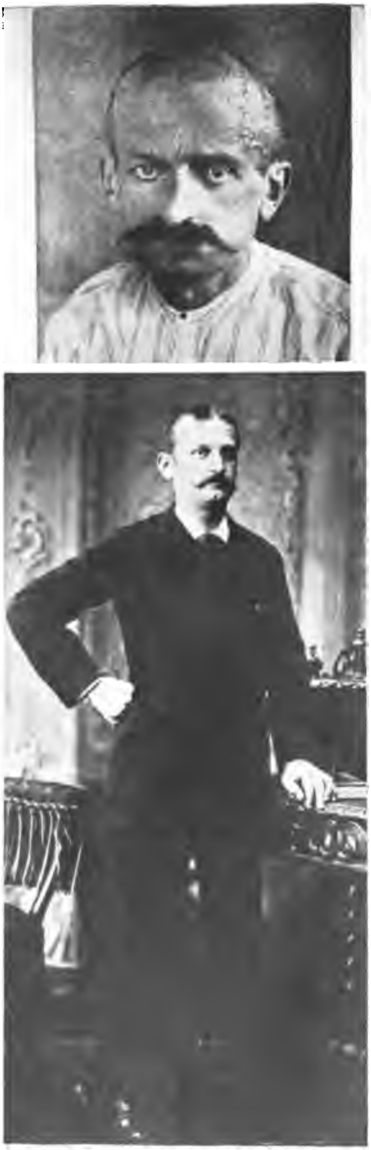

PLATE XVII.

Paget's Disease or Osteitis Deformans. Photograph of the patient in 1893 and 1909.—Pascarolo and Bertolotti. (Page 222.)

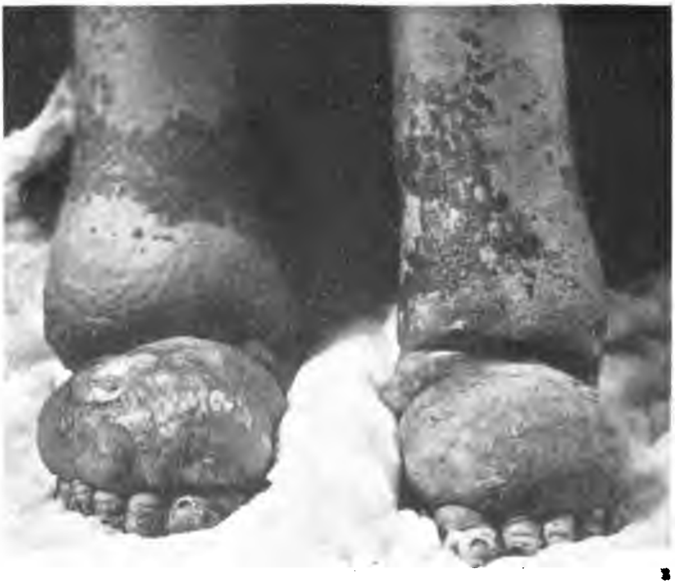

PLATE XX.

A Variety of Acquired Trophedema.—Ramadier and Marchand. (Page 224.)

sisted merely of mental depression with ideas of persecution and hallucinations of hearing. The trophic disturbances present were those seen in dysthyroidism. The edema of the lower extremities can hardly be ascribed to any nosographical type yet defined. The skin of the edematous region is rosy, and not pale as in ordinary trophedema, and is distended by a liquid apparently under the influence of gravity. While trophedema is hard and incompressible, the edema in this case is gelatinous, the very hypertrophic skin seemingly resting upon a bed of soft tissue. The authors believe it to be a variety of trophedema, however, but not like the variety described by Meige and others. (See Practical Medicine Series, 1902, Vol. X, p. 194.)

MENTAL DISEASES.

PSYCHOSES. BIOLOGICAL CLASSIFICATION.

An attempt to classify the various psychoses and neuroses upon a biologic basis is made by Klippel.[1] His paper is an attempt to show that the different somnolent states, the emotional states, and the fatigue states are all functional reactions in a deranged organism. There are thus three great classes or groups of the psychoses and neuroses.

(I) *Somnolent Psychoses.* These include all psychoses in which disorders suggestive of the sleeping state occur, these disorders being sensory, psychic, and motor. For example, when a patient shows simultaneously both hallucinations and catatonia, he is exhibiting symptoms suggestive of the somnolent state,—dreams and physical repose. The general characters of somnolent psychoses comprise absence of attention and control, of attentive reasoning, of astonishment before unforeseen events, absence of doubt as to a matter before further information is gained, and the absence of the sense of identity, time, and place, the three French "unities." These deficits indicate a partial or reduced mental life. Hence there is credulity, suggestibility, automatism, confusion, incoherence, illusions, and hallucinations. The dream state and the delirium of the somnolent psychoses are merely made up of these elements. The somnolent hallucinations are frequently images which move or rather transform themselves before the eyes, so to speak, of the delirious patient. From these, reflexions and ideas arise without coherence, and often the dreamer grafts upon one hallucination a new dream which appears to him like a memory, giving the idea of "false memory." In a dream, at times, the ex-

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(1) La Semaine Méd., July 21, 1909.

traneous factor which is its cause appears as the climax. Thus the sound of a bell, heard by the subconsciousness, excites a dream in the normal sleeper, and at the end of the dream the sound of the bell comes in very pat as its *finale* just before the dreamer awakens. In the somnolent psychoses this same *psychic inversion* occurs, and for example, when the telephone or the bell rings, the patient announces that he was warned in advance, or that spirits told him the bell would ring. It seems as if the original sensation causing the dream is effaced to reappear at the end of the process as its conclusion.

Just as in dreams identity, time, and place are all muddled up at times, so the victims of the somnolent psychoses confound places with relation to time, confound their own identities, double their personalities, confound the persons about them with others, so that illusions are accumulated upon illusions till the confusion is complete.

Sometimes just as a sleeper can be awakened, so can these patients with the somnolent psychoses. For example, a delirious patient, striking a piece of furniture with his fist, believing it to be an animal, is suddenly aroused from his dream by the unexpected contact with the outside world.

Just as amnesia is normal as to our dreams, so it is to be expected in the somnolent psychoses. Hence the discontinuity in the psychic life. Motor disturbances akin to somnambulism also occur, such as ambulatory automatism, catatonia, and catalepsy.

The following forms of somnolent psychoses are differentiated by the author:

1. Hypnagogism, a name applied to that variety of hallucinations which precede complete sleep, and lead up to it.
2. Dreaminess, that is to say an incoherent dream, fragmented, with a troubled sleep-state, loquacity, logorrhea, somewhat resembling the delirium of typhoid fever and other infections.
3. Nightmare type, or the type in which a dream-state is associated with visceral sensations, such as epigastric constriction, respiratory oppression, cyanosis, sweats, zoöpsia, panophobia. The delirium of alcoholics and of autoinfected individuals illustrates the type.

4. The incoherent hallucinatory dream, or the dream-state proper, just as in an ordinary dream.
5. The vision, a more complex dream, more real and connected and plausible.
6. The revery-state, a kind of intellectual dream-state.
7. The motor or somnambulistic dream.
8. Various combinations of any of the above forms.

In general Klippel concludes that the somnolent psychoses, multiplied as they are in form, and various sorts simultaneously appearing in the same individual, are merely functional reactions in a diseased organism. Pathogenic agents, very indefinite in number and very diverse in nature, cause these somnolent psychoses. Hence the variability seen in their intensity, duration, and evolution. The somnolent psychoses are allied with infections, auto-intoxications, accidental or medicamental poisonings, organic psychopathies, delirious seizures in subjects with a nervous taint, and with the psychoneuroses. The psychoses may persist after the cause has passed away.

(II) *Emotional psychoses* are those in which there is involvement of the emotional side of the individual, in its broadest sense. The manifestations of these emotional psychoses may be sensory, motor or psychic. Many tremors, choreiform movements, tics, and spasms are built upon an emotional substratum. Emotion, too, may give rise to a delirium, exactly like a toxic delirium, or it may cause hallucinations with a tendency to obsessions, and various phobias. Disturbances of the affective tone are at bottom emotional psychoses, described under various names, such as delirious obsessions, megalomania, melancholia, mania, fear of suicide or death, or ruin, self-accusations, etc. Besides these mental states, an attentive examination will show a multitude of sensory, motor, and psychomotor, hypo- and hypersecretory, vasomotor, thermic, visceral, and trophic disturbances which accompany the emotions.

Klippel distinguishes the following forms of emotional psychoses: 1. A form in which the cœnesthetic affective state present at the time predominates, being accompanied by affective cœnesthetic hallucinations. This affective state is usually one of immediate joy or sadness, without reference to the future.

2. A form in which predominate conditions of want,

desire or repulsion, summed up in the words desire and fear. Often doubt is joined to these emotional states as an essential element; and doubting fears of untoward future happenings, uncertainty, and suspicion often follow. Indeed doubt often becomes the dominant characteristic in this group of cases.

3. A form distinguished by the predominance of the affective tone in the general reactions of the emotivity, comprising states of exaltation and depression. The speech, features, gestures, attitude, instinctive and voluntary movements of the patient are all significant. Violence, dejection, fury, gaiety, megalomania, suicide, agitation, vanity, anguish, etc., all appear in cases of this form of emotional psychosis.

4. Forms of different degrees of development or evolution of a given sensory, motor, or psychic syndrome.

5. Combined forms; for example, the form of association by succession of the exalted and depressed states.

(III) *Psychoses of Fatigue.* These are numerous and varied. Among them we find a double element which may be expressed in two words: paresthetic asthenia, meaning essentially that a given act is weakly executed, and with perversions of sensibility. These psychoses derive their reactions from profound or visceral sensibility. Hence the patient has great difficulty in explaining the sensations which he feels, and he uses comparisons to define his symptoms. One will meet with such expressions as "It is as if," or "One might say," or "I do not know how to express," or "What I feel is indefinable." The weakness is evidenced by the diminution or ineffectiveness of activity, by the difficulty of sustained effort, by the general diminution of the vegetative functions. The paresthesiæ are such as occur in normal fatigue, and affect the deep sensibilities, localized in the muscles, tendons, aponeuroses, articular ligaments, bones; they are described as aches, heaviness, a feeling of compression, or bruising. The paresthetic sensations are such as to provoke an affective state of lassitude, sadness, or melancholy. The neurasthenic state very well illustrates this group of fatigue psychoses, and all of the paresthesiæ ever found in neurasthenia appear in the various forms of fatigue psychoses.

The various forms of psychoses of fatigue distinguished by Klippel are as follows:

(1) Forms showing certain visceral paresthesiæ more or less localized, causing hypochondriasis, or forms limited to certain organs.

(2) Forms in which paresthesiæ are multiple.

(3) Forms in which the cœnesthetic sensation of weakness gives rise to delirious concepts concerning the physical, intellectual or moral life, to aboulia, to an impossibility to react, to extreme lassitude, general negations, and transformations of the personality.

(4) Forms in which one meets with incoherence and euphoria strangely united with an underlying state of evident weakness.

(5) Forms in which the element of weakness, of torpid asthenia, or of irritable asthenia occur in all varieties, often leading to a delirium, the delirium of exhaustion of the old German psychiatrists.

(6) Forms in which the delirium may be the more marked, the deeper the asthenia.

(7) Forms in which there is a polymorphous delirium, with multiple conceptions of persecution, megalomania, melancholy, these being signs of incoherent weakness.

(8) Forms in which there is a primary mental debility, preceding the delirium.

(9) Forms of dementia, which as it advances, slowly effaces the delirium.

PSYCHOANALYSIS.

Attention is again called to the practical value of Jung's association test in the diagnosis of mental states and hysteria, by Pearce Bailey.[1] The association test long ago known and used became of value when it was discovered by Jung that a lengthening of the time of response gave a clue to the patient's emotional past. "He found that a lengthened interval in one or more reactions in a series of words indicates that the stimulus word has touched upon an emotional complex, or a constellation of ideas which exists independently of volition and which is dominated by an emotion of some kind. This discovery immediately converted the association test into a practical and reasonably speedy method of psychic analysis. The Jung test is primarily individualistic. The stimulus word, like the hunting dog, shows where an emotional complex is lurking. It shows what the subject likes and what he dislikes; where he has suffered and what has pleased him; what he regards as friendly and what as hostile; what he fears and to what he is indifferent. These biographical data come to light without the subject's knowledge and sometimes against his will, and furnish a rational basis for psychotherapy," which latter is treatment by suggestion, explanation, persuasion, and regulation of environmental influences.

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(1) Am. Jour. Med. Sciences, September, 1909.

"This method is as follows: A list of one hundred current words is printed on a pad and so distributed that there is an alternation of nouns and adjectives and every fourth word is a verb. These words are printed in a vertical column, and between every three or four of them a space is left, so that if there seems to be a need of a word not on the printed list, it can be written in. Each word is numbered, so that it can be referred to by number without pronouncing the word. On the same horizontal line as the number and the word three spaces are left, one for the time record, one for the reaction word, and one for the reproduction or the response which is given on the second trial (if made). If the reproduced action is identical with the original reaction, it is indicated (in column three) by the sign +. The method is explained to the patient, and the words are then read to him by a physician, or third person. The association time is taken by a 1/5 second stop watch, and it and the reaction word, or the first word spoken by the subject in response to the stimulus, is entered in the space reserved for it. The second trial, or reproduction, generally necessary for suspicious words only, should be delayed for at least twenty minutes. Failure to reproduce has been shown to be evidence that the stimulus word has touched upon a complex. The test gives its most brilliant results when it brings to light an emotional experience which is no longer a factor in the conscious life of the individual, but which nevertheless has started a chain of persisting symptoms."

The practical workings of the test may be best illustrated by a concrete example. A patient referred to Pearce Bailey as having attacks of *petit mal* gave prompt responses to most words in the list, except the following:

Word	First Response	Reaction-time	Second Response
to threaten	to anger	3.5 seconds	to paralyze
water	bread	4.5 "	milk
trapeze	rope	7.0 "	fall
woods	fear	3.5 "	+
journey	trip,	4.0 "	faint
sympathy	lack of sympathy	4.0 "	+
procession	night	4.0 "	plight
suicide	hell or fire	7.0 "	hell.

"The first four of these reactions had reference to events in childhood, the others to later experience. The reaction *to threaten, to anger, to paralyze,* brought her back to when she was a child of eight. A coachman, having enticed her to his room, took her on his lap, pressed her to him, kissed her, and then threatened to injure her if she ever told. *Water* recalled an experience, also in early childhood, when she saw her brother drowned; *trapeze,* when, also as a child, the rope of a trapeze she was on broke, giving her a bad fall; and *woods,* when she, with other little girls, was badly frightened by being told that Indians were behind the trees. All of these were painful experiences, about which she rarely spoke. Her husband was the only person to whom she had mentioned the affair of the coachman. On the theory that they were concealed complexes which 'mental catharsis' might mitigate, a companion was provided, and to the companion the patient talked every day on these painful subjects. The effect of the free externalization was prompt and pronounced. The patient emerged from a life of invalidism to one of activity; she had no more petit mal attacks."

Valuable, also, is the association test in cases of young adults beneath the surface of whose variegated nervous phenomena lurks a paranoid condition; and also in the differentiation between hysteria and dementia precox. In the latter the complexes are less accessible, more clouded, and in it a larger proportion of the lengthened reaction times will remain unexplained than in a pure psychogenic disorder like hysteria.

Psychoanalysis and psychotherapy were of great value in an interesting case reported by O. Juliusberger.[1] The patient was a woman, 42 years of age, not happily married, suffering with a psychosis which numerous physicians had in vain attempted to cure. In the morning she was nauseated, nearly every day she vomited; she had an especial antipathy toward milk, there were eructations and rumblings, frequent headache, the globus sensation, a feeling of tickling in the throat; often she was hoarse; she complained of weakness, a sense of fatigue, irritability, a marked sensitiveness towards all stimuli, anxiety, depression, moodiness, changeability, insomnia, rushes of blood to the head, hot hands. She thought of all sorts of sad things, knew she could never get well, was afraid she would choke if she ate, having read that a man had choked to death while eating. As a child she had been irritable. At 24 she had married.

Juliusberger was convinced that her whole trouble was due to something troubling her subconsciousness, and by Freud's method endeavored to find out her mental reservations, which she steadfastly refused to divulge. He proposed hypnosis but she energetically refused. She did, however, tell him of two dreams, both of which were indicative of some sexual element at the bottom of her trouble. After much persuasion and after much opposition she confessed that her husband had neglected her, and she was dissatisfied; that he was of a cold nature, even though he treated her very well. She was anxious for a child, and this led her into illicit relations with a friend. In fact by the latter she became pregnant, but miscarried. She then stopped all sexual intercourse with this man, and the sexual act became nauseating. For years she had had no sexual relations with her husband, and they slept in separate rooms. During her illicit relations with her lover her conscience constantly reproached her.

These confessions gave a great insight into her soul life, but it was clear that the roots of the disease were even yet not laid wholly bare. Requested to search her memory further, she recalled that as a little girl of 5 or 6 years of age, she once went into a room where the maid and a male

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(1) Berliner klin. Woch., Feb. 8, 1909.

servant were drinking brandy. She asked for some, and they gave it to her. Then the man took her into a chamber and licked her genitals. This seemed to account for her nausea and vomiting. But there was another factor. The first time she ever had illicit intercourse with her lover was after taking too much alcohol, and she had not undressed prior to the sexual act. And again the second time she had her clothes on. (In one of her dreams she mentioned lying dressed in bed, with a man beside her.)

Her morning nausea was proved to date from another sexual episode. The man, with whom she had her liaison, once remained with her very late, and although she always was in a state of great anxiety every time she met him for fear her husband would return, on this particular occasion she was extraordinarily anxious because the house-door was supposed to be locked and the key could not be found. In fact her lover did not get away until early in the morning.

The disgust for milk was easily explained. The last time her lover came she was lying on a lounge. He opened her dress and sucked her breast, exciting her, and then the pair had sexual intercourse. Thereupon he said: "I got a drop of blood from you." In fact she had just finished menstruating. This incident nauseated her, and sexual relations with him then and there ceased. After all these confessions she no longer feared to be hypnotized, but hypnosis was not now necessary. With the unveiling of the secrets of her life by psycho-analysis, the patient began to improve. "Her relations to her husband were hygienically and ethically regulated," Juliusberger naively remarks, and her whole inner life was guided and directed, while her body was also treated. The result was a cure.

DEMENTIA PRECOX.

The question of the advisability of thyroidectomy in catatonic dementia precox is discussed in a preliminary report by A. B. Kanavel, L. J. Pollack and A. B. Eustace.[1] The following conservative statement precedes the paper: "Before beginning a discussion of this subject, we wish to emphasize that the present contribution is but a prelimi-

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(1) Illinois Medical Journal, September, 1909.

nary report upon which we feel an ultimate decision can not be based. It is but a report of work in progress.

"It has long been thought that certain mental disorders might have some relation to the perverted secretion of these glands. Laignel-Lavastine in an address on psychiatry at the recent congress held at Dijon summarized our knowledge upon this subject and submitted certain questions to the congress for discussion. The consensus of opinion of the congress was that some relationship *probably* existed between disturbances of these organs and certain psychic disorders; and, secondly, that, taking into consideration cerebral predisposition, we may admit the *possibility* of a glandular origin of certain cases of cerebral weakness, dementia precox, partial delirium, functional nervous disorders, hysteria, neurasthenia and psychasthenia.

"Berkley, following the same line of thought, has arrived at the conclusion that there is a causal relationship between dementia precox and the thyroid gland. He bases his assumption upon the following:

"1. He has demonstrated that desiccated thyroid gland and iodin, which is now known to be an essential element in thyroid metabolism, aggravate the symptoms.

"2. The disease often arises at puberty, when the ovaries and thyroid, in common with other organs, take on a change of function.

"3. There is a real or chance similarity between certain symptoms of this disease and exophthalmic goiter. For instance, the increased reflexes, the heightened mechanical muscular irritability, hyperidrosis, tremor, skin changes, profound loss of weight, rapid pulse, and dermographia. At times, the thyroid gland is markedly enlarged, and very frequently there is a fullness in the neck.

"Acting on this assumption Berkley and Follis operated upon eight cases and reported their results in the *American Journal of Insanity* of January, 1909. At the time of operation three patients had been insane and catatonic; five out of the eight recovered completely and remained well for from two to eight months when they were last seen. One of the remaining three was somewhat improved and two showed apparently no result. These last three were all insane over thirteen months when operated upon."

Kanavel, with the assistance of Pollack and Eustace, operated upon ten males and one female, eight being cases of purely catatonic dementia precox, the illness having lasted from under one year to from 5 to 10 years. In each case about seven-eighths of either the right or the left lobe was removed without cutting any muscle except the platysma. The patients all made an immediate and uneventful recovery. In three cases parathyroids were removed by choice. The glands were sectioned and studied, the findings being as follows:

"Two were practically normal, two showed excessive parenchymatous growth; one was a marked colloid goiter, while five showed moderate colloid growth. Marked hyperemia was present in all of the glands and microscopic hemorrhage was commonly present. It is readily admitted that the above classification is not a scientific one, since it is now known that frequently one type shades into the other, but we have chosen the predominating characteristic and it makes a clinical division which is readily understood by all.

"To sum up the matter, one cannot assume any distinct pathological basis for ascribing dementia precox to a hyperthyroidism similar to Graves' disease. We recognize, of course, that this is only one phase of the question and that the pathological picture cannot definitely exclude a perverted thyroid metabolism as a factor. That partial thyroidectomy is absolutely of no avail in old cases I believe it is safe to assume. But when one remembers that Berkley has had five cases apparently completely recover and we have had two greatly improved, if not cured, by this procedure, and nothing else is known which may offer any hope in this group of most pathetic psychoses, it seems justifiable to continue our investigations in the early cases."

General Paresis.

Etiolog Tabes and general paralysis of the insane, in the light of recent investigations by numerous observers must be wholly ascribed to syphilis. Fritz Lesser[1] discusses the subject very thoroughly, affirming that today it is no

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(1) Berliner klin. Woch., Sept. 28, 1908.

longer a question as to the existence of a relation between syphilis and tabes and general paresis, but rather the question as to what that relationship is. L. Michaelis and Lesser investigated 23 paralytics and found 21 positive Wassermann reactions. Of the 2 negatively reacting individuals one was subsequently proved to be suffering from a nonsyphilitic arteriorsclerotic dementia and the other had never been definitely diagnosed as general paresis by Dr. Max Edel. Later 16 more cases were examined, all giving a positive reaction. Plaut in 95 cases obtained a positive reaction every time. We can therefore draw the conclusion that in a suspected paretic a negative reaction with great probability speaks against the diagnosis of general paralysis.

The specific disease begins apparently in the meninges and spreads to the gray matter, causing it to atrophy. Hence in post mortem examinations there are found thickenings of the dura and cortical atrophy. The symptoms of paresis appear only after the cortex has atrophied, and the early stages of the process in the dura give no symptoms. Hence it is not strange that destroyed nerve substance cannot be restored by mercury and iodids. One practical conclusion can, however, be drawn: Every syphilitic should be treated until he fails to show the Wassermann reaction. Since paretics always show the reaction it follows that they were never cured of their syphilis.

Symptomatology. Remissions in general paresis with a report of 3 cases are discussed by M. J. Karpas.[1] True recoveries do not exist, but remissions may occur, being very gradual, reaching their acme perhaps in the course of months. After the mind clears, it may remain so for a varying length of time, but inevitably it swings back into its demented state. The duration of the remission varies considerably from a few months to as much as three or four years, but very rarely longer. The expansive and agitated forms are subject more often to remissions than the depressed or demented types. As a rule remissions are manifest in the early part of the malady before dementia develops. The relation of suppuration and infection to remission in general paralysis has been often observed, and

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(1) New York Med. Jour., July 17, 1909.

Karpas instances many cases from the literature. In a series of 70 carefully studied cases of his own, Karpas has seen only three patients in whom an unquestionable remission of the malady was evidenced, and these three cases are reported at length. Of these two were tabetic cases and one was the pure cerebral form.

"The disease picture, save for the remission, presented no anomalous traits, in each instance the diagnosis of general paralysis could be made without reservation."

Two cases of general paresis of unusually long standing, with post mortem findings are reported by the same author.[1] The usual duration is from two to five years, cases in which the duration is over eight years being exceptionally rare. Of Heilbronner's analysis of 500 paretics, 5 only were of 10 years, and 1 only of 18 years' duration. Binswanger declares that some of his paretics lived over 20 years. The first case reported by Karpas is now living, twelve years after the onset of the disease. Her husband died of general paralysis of the insane. In 1896 the patient's outbreak occurred suddenly, and she became confused, rambling and disconnected in her conversation. Physical examination showed exaggerated knee-jerks, unequal pupils with sluggish reaction to light and accommodation. As time went on the dementia has been very slowly progressive. At present she has an Argyll-Robertson pupil, fibrillary tremor, unequal knee-jerks, and in writing elision of syllables and letters.

The second case is much more convincing, occurring in a woman who died at 58 years of age, having been afflicted eighteen years. In 1890 she became peculiar, seclusive, and depressed. She had a history of having contracted syphilis early in her married life. Placed in a private hospital in March, 1892, she remained until December, and left "apparently doing well." For six months she remained at home and was unnaturally quiet, without motive. Again she went to a private sanatorium for six months. In 1894 she became again abnormally depressed and suicidal and was sent to a hospital for the insane. In October, 1895, there is a record of unequal pupils, tremor of the tongue, ataxic speech, and exaggerated knee-

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(1) New York Med. Jour., March 6, 1909.

jerks. A period of ideas of grandeur followed, and in that same period she became subject to convulsions, which recurred monthly. From 1898 to 1907 the patient's psychosis assumed the form of a quiet dementia, with mild euphoria. Death took place at the Manhattan State Hospital December 17, 1908, and an autopsy was at once performed. Without going into all the finer details of the findings it may be stated that the histopathological picture of the cerebral cortex deviates from that of cases of shorter duration by the scarcity of infiltration and relative increase of glial proliferation and finer blood vessels which show marked tendency to endothelial proliferation.

Amnesic Seizures in general paralysis of the insane are not infrequent according to R. Benon,[1] and he reports three cases. The amnesic ictus is characterized by a sudden, more or less complete loss of memory for a short period of time. The amnesia comes on rapidly, usually abruptly. The patient knows nothing, remembers nothing, inquires as to his whereabouts. In a few moments he comes to himself, but knows nothing of his mental lapse. In the first case, the onset of general paralysis was marked by three amnesic strokes, separated from one another by several days. The patient was not forced to give up work until two years after these seizures. He subsequently died of his disease. In the second case the amnesic ictus chiefly concerned the patient's orientation (topographical agnosia). Amnesia followed progressively, being retro-anterograde and diffuse. In the third case the stroke was several (10-15) minutes in coming on; during the continuance there was motor and verbal automatism. In all of the cases the most salient feature is the disorientation of the patient. The duration of the amnesic status is only ten or fifteen minutes to four or five hours. Amnesic strokes appear to be an acute episode in general paralysis of the insane.

The Juvenile Form. A case of juvenile general paralysis is reported by Rémond and Chevalier-Lavaure[2] in a girl, 14 years of age, a public charge, her parentage being unknown. She was never of much intelligence, but when she reached her fourteenth year her mental activity began to

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(1) Gaz. des Hôp., Oct. 1, 1908.
(2) Le Progrès Méd., May 8, 1909.

wane, she became forgetful, and clumsy. Physical examination showed corneal opacities and Hutchinson-teeth. There was a generalized tremor, and ataxia. Fibrillary tremor of the lips occurred when she attempted to speak, and the tongue showed fibrillary twitches. The reflexes were either abolished or were very much diminished. The pupils were unequal and showed the Argyll-Robertson reaction. the finger-nails showed trophic changes, the hair fell out, and the teeth decayed. Great weakness developed, a sacral bed-sore appeared, diarrhea set in, and she died about two years after the first symptom had been noted.

The autopsy showed an abundant cerebrospinal fluid, a red and congested pia mater, absolutely adhered to the cortex, so that in pulling it off the cortex came with it. The adhesions were most pronounced on the right and at the level of the frontal and parietal areas. There was extreme atrophy of the cortical layer especially in the frontal region. Microscopic examination of the cortical cells showed deformity, chromatolysis, displacement and fragmentation of the nucleus, atrophy of numerous cells, infiltration with leucocytes about the vessels and in the pia mater, proliferation of diffuse neuroglia in the cortex and in the superficial subependymal layers, and the destruction of numerous myelinated superficial fibers. The pathologic diagnosis was therefore a diffuse meningo-encephalitis, which is progressive paralysis.

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